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DESTRUCTION OF THE SPECIALIZED CONDUCTION TISSUE IN THE LIVE CANINE HEART WITH IODINE

J. R. Wennemark, M.D., R. Benvenuto, M.D., and
F. John Lewis, M.D., Chicago, Ill.

IT HAS LONG been known that iodine could be used as a stain in histologic study of high glycogen-containing tissue. However, there has been surprisingly little application of this method in the study of the conductive system of the heart, especially as a macroscopic method of demonstration when applied to the endocardial surface of the heart. In 1885, Marchand[7] applied an iodine solution to the endocardium of the sheep heart and noted the visualization of the conductive system. Much later, Taufic, Lewis, and Bashour[9] in 1957 applied Lugol's solution to the autopsied canine heart to demonstrate the bundle branches during the study of experimental septal defects and Uhley and associates[10] have again emphasized this technique and have applied it to a radioautographic method using radioactive iodine. These are all techniques for study of the dead heart, but recently Allen and associates[1] have suggested that iodine solution be used as a supravital staining method in clinical material during open heart surgery in order to visualize and avoid traumatic injury to the conductive system. We will present evidence to show that this is a dangerous supravital stain because of its damage to the conduction tissue.

In the canine heart, the bundle branch and Purkinje systems are very well developed and lie immediately beneath the endocardium. If looked for carefully, these structures can be seen with the naked eye as white strands of tissue

From the Departments of Medicine and Surgery of Northwestern University Medical School, Chicago 11, Ill.

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contrasted against the background of myocardial tissue. This has been noted previously in the older literature[2, 6] and has also been noted in the beef,[3, 4, 6] sheep,[4, 6] and human[5, 8, 12] hearts. However, in the human heart this is best demonstrated with gross dissection,[5, 8, 13] and the Purkinje system cannot be seen macroscopically.

Because of its high glycogen content, the right and left bundle branch and Purkinje system of the canine heart are easily stained with iodine solution. This stain gives a dark brown to bluish color and is sharply demarcated from the adjacent myocardium. The staining reaction appears several seconds after the local application of iodine solution to the endocardial surface of the heart. The reaction works on both the fresh and the formalin-fixed heart. Fig. 1 shows the right and left bundle branches and Purkinje systems in the canine heart as they appear with this technique. In Fig. 1*A* the well-formed right

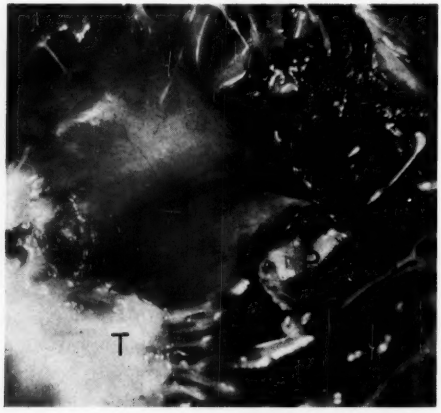

Fig. 1*A*.—Normal right bundle branch of the canine heart stained with Lugol's solution. The main bundle can be seen emerging from beneath the tricuspid valve leaflet (*T*) and bifurcating near the anterior papillary muscle (*P*) into and anterior and posterior radiation.

main bundle branch is seen emerging from beneath the medial cusp of the tricuspid valve, where it originates in the area of the membranous septum. It passes near the muscle of Lancisi and courses down in the area of the septal band of the crista supraventricularis to the anterior papillary muscle where it bifurcates into an anterior and a posterior branch. Minor variations in the pattern of distribution of the fibers in the region of the bundle are noted in some dog hearts in that they may be more diffuse and less easily recognized as a compact bundle. Also fibers may occasionally be seen to branch off the main bundle; however, these have not been seen to end in the Purkinje network. In Fig. 1*B*, the left main bundle branch is seen emerging from the area of the membranous septum. It is very short and diffuse and almost immediately bifurcates into an anterior and a posterior branch.

We have not been able to visualize satisfactorily the bundle of His by this system. However, if the right main bundle is followed up to the membranous septum the common bundle lies immediately in this area.

Several years ago we became interested in this technique as a possible means of visualizing the conduction system in the living heart at the time of open heart surgery in dogs. However, we found that although Lugol's solution stained the conduction tissue well, the staining reaction caused immediate and permanent bundle branch block. Because of this observation, we abandoned the technique for this purpose, but adopted this property of destroying conduction tissue to a study of the mechanism of ventricular activation. The electrophysiologic effect of destruction of the total bundle branch and Purkinje system of the right ventricle of the dog by spraying the endocardium with Lugol's solution has been under study in the dog heart. This technique produces a block which lasts at least 9 months, the longest period of observation we have made.

Fig. 1*B*.—Normal left bundle branch of the canine heart stained with Lugol's solution. The left main bundle is very short and bifurcates almost immediately with an anterior and posterior radiation. The Purkinje network is well visualized.

Stimulated by the report of Allen and associates,[1] we have recently made some further observations as to the effect of iodine solutions on the right main bundle branch.

MATERIALS AND METHODS

Nine medium-sized mongrel dogs were anesthetized with intravenous sodium pentobarbital. A control 12 lead electrocardiogram was taken with a Sanborn Model 150 direct writing machine at a paper speed of 50 mm. per second. Immediately following this, at normothermia, the heart was exposed through a fourth right intercostal incision. The inflow and outflow tracts of the heart were then occluded and, through a right atrial incision, the area of the right main

bundle branch on the ventricular septum was exposed by retraction of the tricuspid valve. In most instances, the main bundle branch could be identified quite well, even before staining. The bundle was stained by applying a cotton pledget dampened with the iodine solution to the bundle area for a few seconds. In all cases the bundle was stained and visualized well. The heart was then closed and the circulation restored. The total time of occlusion was from 2 to 3 minutes. The animal was allowed to recover. An electrocardiogram was taken

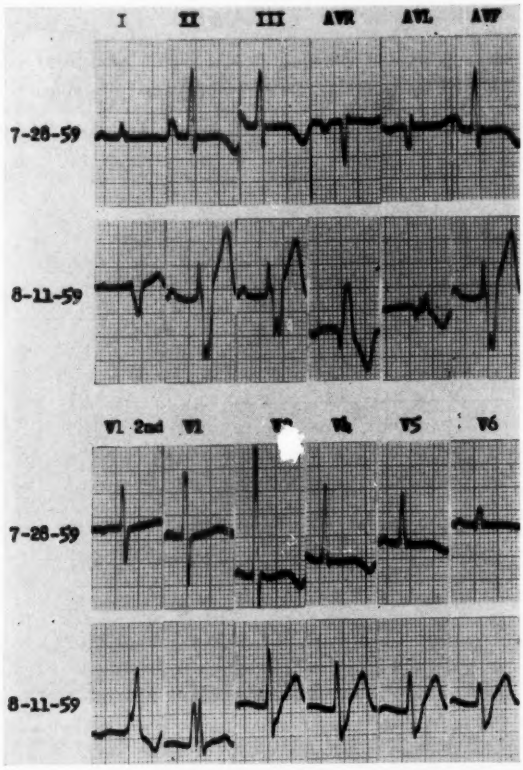

Fig. 2.—Electrocardiogram showing the typical right bundle branch block present 2 weeks following the application of iodine solution to the right bundle branch. The tracing of 7/28/59 is the control before the surgical procedure. The first chest lead is V_1 in the second interspace. V_3 is not taken because of the close proximity of V_2 and V_4 in the dog.

immediately following surgery and again in 14 days, at which time the animal was sacrificed. In 2 cases, just before sacrificing the animal, the left main bundle was injured by a probe introduced into the left ventricle through the posterior myocardial wall near the septum. By this method complete right bundle branch block was proved to be present, for when left bundle branch block was added, complete heart block was noted immediately. The conduction system was examined grossly in all dogs after sacrifice with excess Nembutal. Histologic examination was also performed in 2 dogs.

For the histologic work, a block was taken of the ventricular septum in the area of the bundle and serial sections were made of the entire block, staining alternate sections with the periodic acid-Schiff stain and hematoxylin and eosin. In each heart a block of the left main bundle was prepared in a similar way as a control.

In this study the iodine solutions used were: 5 per cent elemental iodine and 10 per cent KI in aqueous solution (Lugol's solution) in 6 cases, and 5 per cent elemental iodine and 10 per cent NaI in aqueous solution in 3 cases. The latter solution was the one used by Allen and his co-workers.[1] We used it in the above cases only to rule out the possibility of potassium effect with the Lugol's solution.

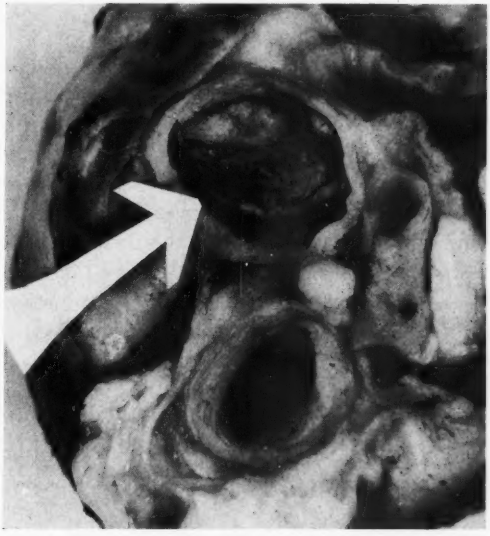

Fig. 3.—View showing almost complete occlusion of the pulmonary artery by a thrombus originating from the area of application of Lugol's solution to the septal endocardium.

RESULTS

All 9 animals showed a normal control electrocardiogram. There was complete right bundle branch block in the tracing immediately following surgery and in the 14-day tracing in 8 cases, while in 1 case, in which Lugol's solution was used, the block appeared to be incomplete. Fig. 2 shows a typical control and final electrocardiogram. In the 2 cases in which the left main bundle branch was injured, a complete heart block developed, which proved that the block in the stained right bundle was complete.

At autopsy examination, a severe tissue reaction was seen in the area of application of the iodine solution in 7 cases. In 2 cases this area was covered with a large thrombus. In 1 case (Fig. 3), the thrombus extended into the pulmonary outflow tract and proximal main pulmonary artery, causing a severe

degree of obstruction of this vessel. In 2 cases the endocardial tissue reaction was not as severe and the main bundle branch could be seen as a dense white band in the area stained, which was a distinct change from its normal appearance. In all cases there was a loss of ability of the bundle to stain with Lugol's solution in the area involved. Distally the uninvolved bundle and Purkinje tissue retained this property, an observation which we have also made following cutting of the bundle.[11] There appeared to be no difference in the effect of the two solutions used.

Examination of the involved area in the 2 cases studied histologically revealed the endocardium to be inflamed and fibrotic. The conduction tissue could not be identified with either the periodic acid-Schiff or hematoxylin and eosin stain. Some sections showed organizing fibrin on the surface of the endocardium.

DISCUSSION

The use of a supravital stain to give good visualization of the conduction system during open heart surgery would be desirable in order to prevent injury to the system during the procedure. Allen and associates[1] have recently advocated the use of an iodine solution (5 per cent elemental iodine and 10 per cent NaI in aqueous solution) to accomplish this in experimental and clinical work, but our experience, as presented, clearly shows that iodine solutions are dangerous because of their deleterious effect on the conduction tissue. This is manifested as both functional and histologic change. Also, there is a severe endocardial reaction and sometimes thrombus formation in the area to which the solution is applied.

At the present time we do not know of any supravital stain which can be used on the conduction system. However, further investigation is desirable in this field because of the frequency of conduction disturbances following open heart procedures.

The bundle branches, especially the right, can be seen sufficiently well with the naked eye in the canine heart to be of use in experimental open heart procedures when injury to the system is to be avoided.

Iodine solutions give very good contrast of the bundle branches and Purkinje tissues in the autopsied canine heart for purposes of gross morphologic study.

SUMMARY

1. Iodine solutions were shown to stain selectively the bundle branch and Purkinje system of the dog heart due to the high glycogen content of these structures.

2. Iodine solutions were applied to the right bundle branch of the canine heart in 9 living animals in order to evaluate this as a supravital staining method.

3. This procedure produced bundle branch block in all cases and subsequent gross examination and histologic examination in 2 cases showed severe damage to the conduction tissue.

4. The use of iodine solutions to supravitally stain conduction tissue in the human being should be thoroughly evaluated because of the readily demonstrable and permanent damage this substance produces in the conduction system of the dog.

We are grateful to Dr. Robert B. Jennings of the Department of Pathology of Northwestern University Medical School for help in evaluating the pathologic material.

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PRIMARY CARCINOMA OF THE LUNG

A Follow-Up Study Including Pulmonary Function Studies of Long-Term Survivors

John C. Jones, M.D., Joseph L. Robinson, M.D., B. W. Meyer, M.D., and Hurley L. Motley, M.D., Los Angeles, Calif.

THE PURPOSE of this paper is twofold: first, to present data concerning the fate of 513 people operated upon for primary carcinoma of the lung between July 1, 1942, and Jan. 1, 1959; and, second, to call attention to the problem of cardiopulmonary illness faced by those long-term survivors who were successfully treated by pneumonectomy.

AGE

Patients varied in age from 32 to 80 years, with the highest concentration lying in the sixth decade, or more specifically, between the ages of 55 and 60 years (Table I). We have had less success in treating patients under 40 and over 70 years than those in other age groups. Of 9 patients under 40, only 2 had lesions which permitted pulmonary resection. One of these died within 3 months of the operation from metastases, and the other, although living a year and a half after surgery, has had a recurrence and is being treated by radiation therapy. Those at the other end of the age spectrum also did poorly, but for a different reason. There were 35 patients 70 or more years of age for whom we did a thoracotomy, and in this group the resectability rate of 51.4 per cent was almost as good as the over-all rate of 53.3 per cent. But the surgical mortality was considerably higher, being 8.6 per cent as compared to an over-all 2.9 per cent. There were 3 deaths, all occurring among the 9 patients who had a pneumonectomy, for a surgical mortality rate for this procedure of 33.3 per cent. There were no surgical deaths among the 16 patients explored, nor among the 10 who had lobectomy. In our experience, a deficient cardiopulmonary reserve is the greatest hazard to these older patients, and we now prefer to have complete pulmonary function studies done on them prior to surgery. We try to resect the amount of lung tissue which will provide the patient with the best over-all prognosis, taking into consideration both the cardiopulmonary function, and the nature and extent of the neoplasm.

From the Departments of Surgery and Medicine and the Cardio-Respiratory Laboratory of the University of Southern California School of Medicine.

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TABLE I. AGE DISTRIBUTION IN 513 THORACOTOMIES

AGE	LOBECTOMY	PNEUMONECTOMY	ALL RESECTIONS	EXPLORATIONS	TOTAL THORACOTOMIES
Under 40	2	0	2	7	9
40-44	4	12	16	20	36
45-49	5	28	33	29	62
50-54	8	38	46	38	84
55-59	25	47	72	62	134
60-64	19	45	64	36	100
65-69	14	18	32	21	53
70 and over	10	9	19	16	35
Totals	87	197	284	229	513

SEX

Among 513 patients were 68 (13.3 per cent) females. The proportion of females in our series increased slightly in the latter half, as compared to the first half. Up to the end of 1950, 12.2 per cent of 196 patients were women, as compared to 13.9 per cent of 317 patients from the end of 1950 to the end of 1958. This increase of 1.7 per cent should not necessarily be taken to mean that there has been a similar increase in the amount of pulmonary carcinoma in women. An analysis of our data indicates that this increase is due largely to the greater number of asymptomatic, peripheral lesions being found with the more frequent use of routine chest x-ray examinations. Comparing resectability rates in the two periods, we see that in males the rate increased less than 2 per cent (from 54.7 per cent to 56.4 per cent), while the rate for females jumped over 30 per cent (from 33.3 per cent to 63.6 per cent) (Table II). Also the proportion of resections, which was limited to a lobectomy, rose from about 10 per cent in the early period to about 42 per cent in the latter period. Likewise, the proportion of adenocarcinoma in the resected specimens rose from 12.8 per cent to 18.1 per cent (Table III). All of these facts, we believe, point to the conclusion that the earlier diagnosis of peripheral lesions has been of greater significance, proportionately, in females than in males.

TABLE II. RESECTABILITY RATES BEFORE AND SINCE JAN. 1, 1951
SEX COMPARISONS (513 THORACOTOMIES)

	BEFORE JAN. 1, 1951			SINCE JAN. 1, 1951			TOTALS		
	MALE	FEMALE	TOTAL	MALE	FEMALE	TOTAL	MALE	FEMALE	TOTAL
Thoracotomies	172	24	196	273	44	317	445	68	513
Explorations	78	16	94	119	16	135	197	32	229
Resections	94	8	102	154	28	182	248	36	284
Resectability rate	54.7	33.3	52.0	56.4	63.6	57.4	55.7	53.0	55.4

TABLE III. PERCENTAGE ADENOCARCINOMA IN PATHOLOGY SPECIMEN
284 RESECTIONS: 248 MALES—36 FEMALES

	BEFORE JAN. 1, 1951	SINCE JAN. 1, 1951	TOTALS
Males	11—11.8%	19—12.3%	30—12.1%
Females	2—25 %	14—50 %	16—44.4%
Totals	13—12.8%	33—18.1%	46—16.2%

The early surgical results are shown in Table IV. Among the 513 patients having thoracotomy, there were fifteen deaths within the first 2 weeks, for a surgical mortality rate of 2.9 per cent. Only one death in 229 operations (0.4 per cent) occurred following exploration. Likewise, following 87 lobectomies, there was only one death, for a surgical mortality rate of 1.1 per cent. The highest rate occurred in those patients having pneumonectomy, where there were 13 deaths in 197 operations, for a mortality rate of 6.6 per cent. This gives an over-all mortality rate among 284 patients having pulmonary resection of slightly under 5 per cent (14 deaths). Three of these 14 deaths occurred in poor-risk patients over 70 years of age who had pneumonectomy.

TABLE IV. EARLY SURGICAL RESULTS (513 THORACOTOMIES)

	MALES	FEMALES	TOTAL
Number of thoracotomies	445	68	513
Surgical mortality	15	0	15—2.9%
Number of explorations	197	32	229
Surgical mortality	1	0	1—0.4%
Number of resections	248	36	284
Surgical mortality	14	0	14—4.9%
Number of lobectomies	65	22	87
Surgical mortality	1	0	1—1.1+%
Number of pneumonectomies	183	14	197
Surgical mortality	13	0	13—6.6%

LATE RESULTS OF SURGERY

For about one half of our patients who had pulmonary resection, the operation had little influence in prolonging their lives (Table V). These, obviously, were the patients in whom the carcinoma had extended beyond the lung or had metastasized by way of the blood or lymph stream prior to the time of surgery.

TABLE V. RESECTION SURGERY: LATE RESULTS

YEARS IN WHICH RESECTED	1942-1957	1942-1955	1942-1953	1942-1948
Length of time observed	1 year	3 years	5 years	10 years
Number of patients	249	202	162	64
Number dead or lost	123	139	126	52
Per cent dead or lost	49.4	71.8	80.9	81.2
Number known alive	126	57	31	12
Per cent known alive	50.6	28.2	19.1	18.8

In a few of them, there was suggestive evidence of metastasis or extension at the time of surgery and the prognosis was thought to be bad. But in most of them there was no apparent evidence of metastasis at the time of resection. Approximately 50 per cent of those having a resection died within the first year after surgery, which compares with a 95 per cent death rate within the first year after exploration for a group followed earlier.[1] After the first year, the death rate for carcinoma rapidly diminished, and by the third postoperative year, about 90 per cent of those who were to die of carcinoma had already done so (Fig. 1).

In previous publications,[1, 2] we presented follow-up data for a small group of patients who had been treated by lobectomy. Lobectomy had been done in

these patients as an operation of choice, the criteria for the selection being (1) a peripheral lesion well removed from fissures, pleural surface, and point of bronchial transection, and (2) in which the mediastinal and hilar lymph nodes did not appear to be involved in a metastatic process. This small group of patients had a 3-year survival rate of 75 per cent. Since that time, we have performed lobectomy in some patients as a palliative procedure, and in others as a second choice because of poor cardiopulmonary reserve. This has, of necessity, lowered the long-term survival rate for this operation. At the present time, the 3-year survival rate for 52 patients having lobectomy is 51.9 per cent, as compared with 20 per cent for 150 patients having pneumonectomy (Fig. 2).

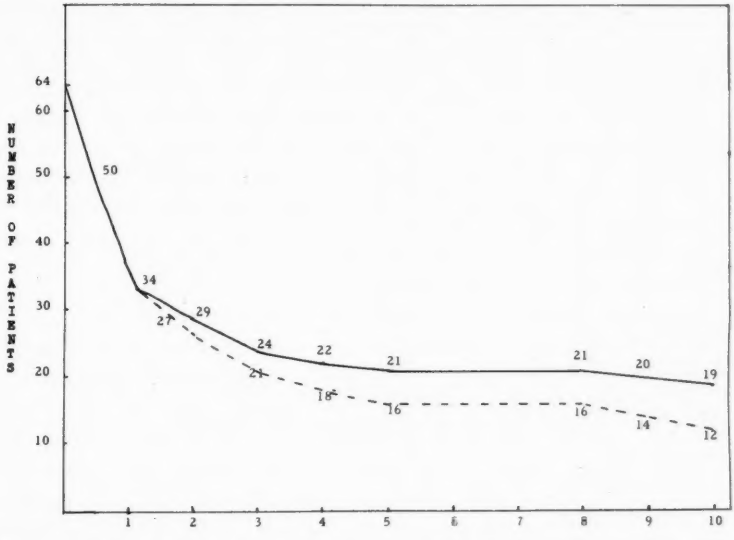

Fig. 1.—Ten-year follow-up after resection on 64 patients—63 pneumonectomies and 1 lobectomy. *Dotted line:* number of survivors. *Solid line:* number of carcinoma survivors.

Ten-Year Survival.—In this series of 513 patients, are 63 who had a pneumonectomy more than 10 years ago. Thirty of these 63 died of carcinoma within the first year after surgery. In the next 9 years, 22 more died; but of the 22, 7, or about one third, died of causes other than carcinoma. Considering the age of the people involved, this is not surprising, nor, perhaps, is it surprising that 5 of these deaths were reported as of cardiac origin. We have wondered, however, to what extent and in what way the loss of one lung may have contributed to their so-called "cardiac" deaths. Were they not in reality cardiopulmonary deaths? And if so, could something perhaps have been done to prolong their lives? It has been suggested that the distention of the remaining lung following pneumonectomy produces pulmonary emphysema, which in turn reduces the cardiorespiratory function. It has been further suggested that thoracoplasty should be done in order to reduce distention in the remaining lung.

In order to study this problem, we decided to bring in the 11 patients who are still alive 10 or more years after pneumonectomy. The length of their survival varies between 10 and 16 years, with the average being 12.7 years. With one exception, the 11 patients are all leading satisfactory lives. They are either working, or are exerting an equivalent amount of physical activity. The one exception is F. O. H., who, strangely enough, is the oldest in respect to survival time (16 years), but the youngest in actual age. At the time of surgery, he was 42 years old. Within the past 2 years, he has had two episodes of right heart failure, and was almost totally incapacitated at the time of the study. He has recently died of right heart failure.

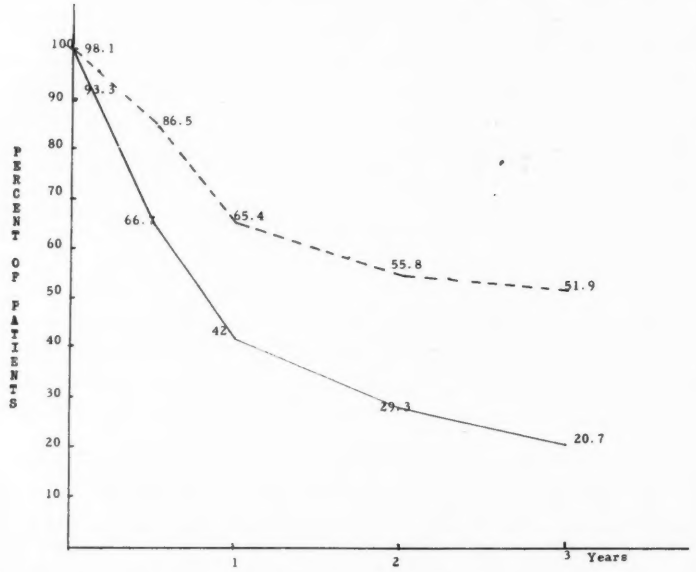

Fig. 2.—Three-year follow-up after resection. *Dotted line:* patients having lobectomy. *Solid line:* patients having pneumonectomy.

We were able to do complete pulmonary function studies on 9 of the 11 patients (Table VI).

TABLE VI. PATIENTS STUDIED 10 OR MORE YEARS AFTER PNEUMONECTOMY

PATIENT	SIDE	AGE	YEARS SINCE SURGERY
1. F. O. H.	Left	57	16
2. H. J. H.	Right	68	10
3. H. B.	Right	65	12
4. H. H.	Right	60	13
5. L. Z.	Left	64	15
6. R. B. (Mrs.)	Right	57	10
7. J. C.	Left	66	12
8. J. O. R.	Left	65	12
9. R. B.	Right	65	14

PULMONARY FUNCTION STUDIES

Methods.—The lung volume measurements consisted of spirograms on the 13.5 liter respirometer before and after bronchodilator drugs such as Vaponefrin and Isuprel. The measurements from the spirogram included total vital capacity, timed vital capacity for 3 seconds, and the maximal breathing capacity. The shape of the spirogram tracing was also of significance. The residual air was measured and checked both by the oxygen open circuit[3] and the helium closed circuit methods.[4] The alveolar nitrogen was obtained on oxygen breathing after the end tidal nitrogen was reduced to 1 per cent as monitored by the nitrogen meter. The oxygen content, oxygen capacity, CO_2 content and pH were obtained on resting and exercise samples of the arterial blood, employing the manometric Van Slyke apparatus. The arterial blood oxygen saturation was determined both from oxygen content and capacity measurements and by employing the Waters double-scale cuvette oximeter for blood. The oxygen capacity was checked both by the flask and Van Slyke methods. Arterial blood samples were obtained at rest and with exercise, on air and graded levels of high oxygen breathing (33.8 per cent and 100 per cent), and with intermittent positive pressure breathing on compressed air. The subject was allowed to breathe the 33.8 per cent oxygen for 8 to 10 minutes before the exercise was started. The pulmonary ventilation measurements were obtained from gas analysis, using the Scholander apparatus, of the expired air collected in a Tissot gasometer. The effective tidal air was calculated from the expired pCO_2 and the arterial pCO_2, the latter measurement by the direct bubble tension technique.[5] The carbon monoxide diffusion capacity was measured by the steady state method described by Filley and his co-workers.[6]

Results.—The lung volume measurements reveal that the total vital capacity in all patients except one was 44 per cent or greater, of the predicted normal for both lungs (Table VII). The normal division of function in the lung is 55 per cent for the right and 45 per cent for the left. The timed vital capacity for 3 seconds was significantly decreased as compared to the total vital capacity in 7 cases. There was a marked decrease in the maximal breathing capacity in 5 cases with a moderate decrease in the other 4, but a significant degree of bronchospasm was observed in all.

The absolute volume of the residual air was markedly increased in Case 1, slightly increased in Case 3, and decreased below the predicted normal in all of the others. In Cases 5 and 6, the residual air was decreased almost in proportion to the vital capacity. The total lung capacity varied from 47 to 68 per cent of normal predicted. The ratio of the residual air to total lung capacity was markedly increased in Case 1, moderately increased in Cases 3, 7, and 9, slightly increased in Case 8, and normal in the other 4. However, the helium equilibration time, the nitrogen washout curves (Fig. 3), the alveolar nitrogen per cent after oxygen breathing, and the absolute volume of the residual air indicates that emphysema was not present in a significant degree in 8 of the 9 patients. A severe degree of emphysema was present in 1 patient

TABLE VII. PULMONARY FUNCTION STUDIES IN PNEUMONECTOMY

	CASE NUMBER								
	1	2	3	4	5	6	7	8	9
Pneumonectomy, right or left	Lt.	Rt.	Rt.	Rt.	Lt.	Rt.	Lt.	Lt.	Rt.
Duration (years)	15	10	12	13	15	10	12	12	14
Age (years)	57	68	65	60	64	57	66	65	65
Height (cm.)	175	167	175	180	181	158	170	173	175
Body surface area (sq. M.)	1.69	1.77	1.85	2.24	1.97	1.71	1.87	1.81	1.96
Vital capacity, supine (ml.)									
Observed	1,615	2,283	1,985	1,874	1,884	1,345	1,813	1,923	1,954
% Predicted	37.9	64.1	49.9	44.4	44.3	44.6	48.7	49.6	49.1
Timed vital capacity (3 sec. ml.)									
Observed	1,071	1,317	1,591	1,415	1,821	1,278	1,481	1,547	1,643
% Predicted	25.1	37.0	40.0	33.5	42.8	42.4	39.8	39.9	41.3
Maximal breathing capacity (L./min.)									
Observed	17.3	21.8	34.2	53.7	68.6	44.6	30.5	48.8	60.3
% Predicted	14.9	19.6	28.7	35.8	53.6	47.9	25.5	41.8	47.7
Maximal breathing capacity after bronchodilator									
% Predicted	18.2	34.8	36.1	37.4	61.2	55.9	32.4	53.0	58.1
Alveolar N_2 after oxygen	1.85	1.31	1.90	0.29	0.66	1.30	1.50	1.35	1.15
Time (min.)	10.0	7.0	7.0	7.0	7.0	6.0	5.0	7.0	6.0
Residual air (ml.)									
Observed	1,932	1,114	1,880	947	1,075	752	1,394	1,318	1,560
% Predicted	136	73.0	110	52.3	58.9	74.8	87.3	79.3	91.5
Total lung capacity (ml.)									
Observed	3,547	3,397	3,865	2,821	2,958	2,097	3,207	3,241	3,514
% Predicted	62.4	66.7	68.0	46.8	48.7	52.2	60.3	58.5	61.9
Residual % of total lung capacity	54.5	32.8	48.6	33.6	36.3	35.9	43.5	40.7	44.4
Ventilation factor (%)*	28.6	49.4	43.5	52.9	59.7	53.3	44.8	51.8	52.2
Arterial Blood Oxygen Saturation (%) Air breathing									
Rest	89.1	92.5	93.7	94.6	91.8	92.3	94.4	92.7	94.1
Exercise	89.5	84.5	93.8	90.4	91.8	92.5	91.3	91.0	95.0
33.8% O_2 breathing									
Rest	96.8	97.4	97.8	97.8	98.5		96.8	96.0	–
Exercise	94.3	92.4	96.7	93.5	97.0	93.4	95.5	94.4	–
IPPB compressed air (rest)	93.8	96.5	97.4	96.5	97.7	96.4	96.5	95.6	96.8
100% Oxygen (rest)	100.0	100.0	99.8	99.4	99.2	97.8	98.5	98.0	99.0
Carbon monoxide diffusing capacity c.c. of CO uptake per min./ alveolar pCO mm. Hg									
Rest	4.41	3.62	5.64	5.61	5.41	6.06	15.37	7.95	6.31
Exercise	5.99	7.42	10.00	14.01	13.23	12.24	18.67	15.18	24.95
Arterial blood CO_2 cont. vol. %									
Rest	54.7	51.8	54.8	48.2	54.5	54.0	50.9	50.1	54.8
Exercise	51.2	50.4	51.2	45.8	50.3	52.3	49.5	47.6	53.2
Arterial blood pH									
Rest	7.40	7.43	7.42	7.42	7.46	7.43	7.44	7.44	7.41
Exercise	7.40	7.43	7.44	7.41	7.44	7.43	7.42	7.42	7.44
Minute ventilation (L./min./ sq. M./BSA)									
Rest	3.57	3.66	3.92	3.11	3.29	3.34	3.84	4.20	3.82
Exercise	7.71	8.87	8.26	11.2	9.17	7.77	7.48	9.63	10.48

TABLE VII—CONT'D

	CASE NUMBER								
	1	2	3	4	5	6	7	8	9
Tidal volume (ml.)									
Rest	299	399	522	616	332	371	428	631	353
Exercise	420	650	612	738	623	578	538	830	514
Oxygen uptake (ml./min./sq. M./ BSA)									
Rest	100	107	98	123	108	100	98	141	129
Exercise	266	392	333	430	342	350	372	472	438
Oxygen % extracted from inspired air breathed									
Rest	2.81	2.94	2.50	3.97	3.27	3.00	2.55	3.29	3.39
Exercise	3.45	4.44	4.03	3.84	3.73	4.51	4.98	4.90	4.18
Direct resting tension measurements mm. Hg									
Arterial pCO_2	49	39	47	42	42	41	42	40	45
Arterial pO_2	63	82	83	88	80	90	83	87	82
Alveolar pO_2	85	101	95	97	91	93	96	96	91
Inspired-Alveolar pO_2 Difference mm. Hg (rest)	64	48	55	51	57	55	52	52	57
Alveolar-Arterial pO_2 Difference mm. Hg (rest)	22	19	12	9	11	3	13	9	9
Physiological Dead Space Rest ml.	142	164	290	215	153	176	217	288	142
Effective tidal air Rest %	53	59	44	65	54	53	49	54	60
Heart rate (per min.)									
Rest	88	80	60	80	84	68	88	68	79
Exercise	100	120	100	130	144	112	128	108	112
Respiration rate (per min.)									
Rest	20	16	14	11	20	15	17	12	21
Exercise	31	24	25	34	29	22	26	21	40
Basal metabolic rate Per cent predicted	−19.8	−10.1	−17.0	−0.1	−12.4	−13.0	−17.8	+17.4	+7.8

(Case 1) as shown by: (1) the spirogram tracing with marked prolongation of exhalation, (2) the very marked decrease in the maximal breathing capacity, (3) the marked impairment in air distribution in the lung, as shown by the nitrogen washout curve, (4) the marked increase in the absolute volume of the residual air, and (5) the marked increase in the ratio of the residual per cent of total lung capacity.

The uniformity of air distribution in the lung was evaluated by the helium equilibration time, the alveolar nitrogen per cent on oxygen breathing, and the nitrogen washout curve on oxygen breathing with the nitrogen meter. The helium equilibration time was markedly prolonged in Case 1, very slightly prolonged in Cases 2 and 3, and normal in the others. The alveolar nitrogen on oxygen breathing was normal in 7 patients and only slightly prolonged in 2 (Cases 1 and 3). The nitrogen washout curve with the nitrogen meter, obtained by continuous recording of each breath on oxygen breathing, revealed normal washout curves in 4 subjects to an end tidal of 1 per cent in 5 minutes

or less (Fig. 3). In 4 patients, the nitrogen washout was very slightly prolonged beyond the 5-minute time interval, but was significantly prolonged in Case 1 only; and even in this patient the prolongation was not as marked as seen in patients with comparable severe emphysema. A composite graph is shown in Fig. 3 of 36 cases, all with severe emphysema and residual ratios of 50 to 60 per cent of total lung capacity.

The arterial blood studies revealed that only the patient with emphysema (Case 1) had a severe degree of arterial blood oxygen unsaturation at rest (below 90 per cent). A slight to moderate degree of arterial unsaturation was present in the others at rest. With step-up exercises, 1 patient (Case 2) with severe bronchospasm developed a severe degree of arterial blood oxygen

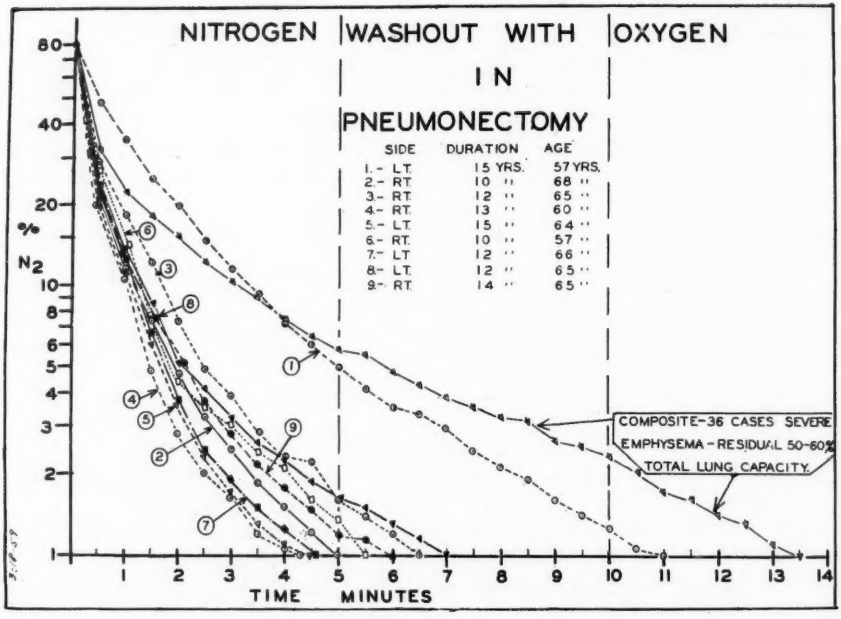
NITROGEN WASHOUT WITH OXYGEN
IN
PNEUMONECTOMY
SIDE DURATION AGE
1.- LT. 15 YRS. 57 YRS.
2.- RT. 10 " 68 "
3.- RT. 12 " 65 "
4.- RT. 13 " 60 "
5.- LT. 15 " 64 "
6.- RT. 10 " 57 "
7.- LT 12 " 66 "
8.- LT. 12 " 65 "
9.- RT. 14 " 65 "
% N2
TIME MINUTES
COMPOSITE-36 CASES SEVERE EMPHYSEMA-RESIDUAL 50-60% TOTAL LUNG CAPACITY

Fig. 3.—Nitrogen washout with oxygen breathing with the Waters nitrogen meter in 9 cases of pneumonectomy and a composite curve of 36 cases of severe emphysema (all with a residual ratio to total lung capacity between 50 and 60 per cent). The nitrogen concentration as per cent in the expired air is plotted on the ordinate against time at 30-second intervals on the abscissae using semilog paper. The nitrogen washout curve was normal for Cases 2, 4, 5, and 7. In Cases 3, 6, 8, and 9, the nitrogen washout curve was very slightly prolonged beyond 5 minutes to an end tidal of 1 per cent and indicates the absence of a significant degree of impairment in air distribution. The nitrogen washout curve for Case 1 was significantly prolonged and characteristic of the type seen in severe emphysema. However, the nitrogen washout for Case 1 was significantly bet'er than the composite curve for severe emphysema of a comparable degree as based on the residual per cent of total lung capacity.

unsaturation and a marked drop below the resting level. In 5 patients, there was no significant change with exercise, and in the other 3, a slight drop in 1 and a moderate drop in 2. The arterial saturation was significantly increased in all patients at rest on intermittent positive pressure breathing on compressed air only, indicating the presence of poorly ventilated alveoli as responsible

for part of the unsaturation. However, part of the hypoxia was due to the perfusion of blood through nonventilated areas with exercise, as the saturation did not increase to 97 per cent or more in all cases on a 33.8 per cent oxygen breathing with an increase in the inspired pO_2 over 90 mm. Hg above that of air breathing.[7]

The carbon monoxide diffusion capacity measurement using the steady state method of Filley and his associates was low at rest in all cases except 1, and with exercise in all cases except 3. The lowest values with exercise were present in the patient with emphysema and the patient with severe bronchospasm (Cases 1 and 2).

The carbon dioxide content of the arterial blood was very slightly elevated above the average normal of 48.5 volume per cent, both at rest and with exercise. The arterial pH was normal both at rest and with exercise. Direct tension measurements of the arterial blood at rest reveal fairly normal values except for the emphysema patient, where the pCO_2 was increased and the pO_2 decreased. The oxygen transport across the lung was also fairly normal as measured by the inspired-alveolar pO_2 difference and the alveolar-arterial pO_2 difference, except for the patient with emphysema (Case 1), and the increased alveolar-arterial pO_2 difference in Case 2 (severe bronchospasm).

The minute ventilation was essentially normal both at rest and with exercise. The resting oxygen uptake was decreased slightly in all patients except 2, as shown by the direct measurement of the basal metabolic rate on air breathing. The exercise oxygen uptake was very markedly decreased in the patient with emphysema (266 ml. per min. per sq. M., BSA; normal 500 to 600). A marked decrease in exercise oxygen uptake was present in 5 patients and a moderate decrease in the other 3. The decrease in the exercise oxygen uptake is quite significant in this series of cases as it indicates the inability to increase the pulmonary blood flow corresponding to the degree of exercise given, and indirectly indicates increased pulmonary vascular resistance and the probable presence of cor pulmonale. There is a slight to moderate degree of impairment in the lung ventilation efficiency, as measured by the per cent of oxygen extracted both at rest and with exercise. The effective tidal air based on the expired pCO_2 and the arterial pCO_2 was significantly decreased in all patients, being below the normal predicted value of 75 per cent. The calculated physiological dead space was markedly increased in 2 patients while at rest.

The data on these 9 patients with pneumonectomy indicate that, over-all, cor pulmonale is a more significant factor than emphysema in producing disability. Except in one instance, emphysema was not a significant factor in these studies of lung function. Air distribution in the lung was normal for 4 patients, very slightly impaired in 4, and severely impaired in the patient with emphysema. The impairment in the arterial blood oxygen saturation was due to poorly ventilated alveoli and the perfusion of blood through nonventilated alveoli, especially with exercise. Bronchospasm and fibrosis appear to be primarily responsible for the hypoxia. The exercise oxygen uptake was significantly decreased in all cases, and to a marked degree

in all cases except 3, where the decrease was of a moderate degree. These measurements indicate the inability to expand the pulmonary vascular bed in a normal manner with exercise and the presence of increased pulmonary vascular resistance. The changes in pulmonary vascular resistance cannot be explained on the basis of pulmonary emphysema in 8 of the 9 cases. Since the rationale of a prophylactic thoracoplasty as performed shortly after a pneumonectomy is to prevent the development of emphysema, it would appear that in these patients such a procedure would have been without value, as significant emphysema is not present 10 to 15 years later. Furthermore, the deformity of a thoracoplasty could have impaired still more the ventilatory and circulatory defects which exist at the present time.[8]

DISCUSSION

The technique of pulmonary resection for carcinoma of the lung is well and long established, and the surgical risk of the operative procedure per se is satisfactorily low. But that is not equally true of the effects of resection upon the patient's physiology. His survival, both short and long time, will depend not only upon the success of the surgeon in resecting all of the neoplastic disease, but also upon the degree of disturbance in his cardiopulmonary system produced by the resection. Since death is the ultimate fate of all mankind, our aim in treating a patient with carcinoma is not only to alleviate suffering, but also to prolong life to the best of our ability.

Our success in arresting the fatal course of a neoplastic process by surgical extirpation certainly leaves a great deal to be desired. In only about one half of those in whom we think we have performed a successful resection will one year of postsurgical life be realized, and only about one fourth of them will survive for a period of 5 years. In our experience, this appears to be true regardless of whether the extent of the resection is a lobe or the entire lung, so long as all visible neoplastic tissue is resected, together with a safe margin separating the resected from the uninvolved tissue. Such being the case, we feel hardly justified in performing a complete pneumonectomy in every patient, regardless of the state of his cardiopulmonary physiology. Any further improvement in salvage rates, so far as surgical treatment of carcinoma is concerned, would appear to depend primarily upon earlier diagnosis and treatment. This is not true in regard to improvement in the survival rates of patients who have had successful resection. Here, there are two avenues for improvement. The first has to do with a better selection of the surgical procedure to fit the patient, and the second deals with reducing factors which are responsible for cardiopulmonary failure following resection. The means to the first are pretty well at hand—that is, the preoperative use of pulmonary function studies and other tests by which physiological status may be evaluated. The attainment of the second objective will depend largely upon the accumulation of additional data.

We think that these studies on this small group of patients have been informative, and that additional fields for investigation are quite clearly indicated. All 9 patients were, at the time of the examination, suffering from

some degree of cardiopulmonary incapacity. In only 1 case was this due chiefly to pulmonary emphysema. In the other 8 cases the disability was obviously due to other factors. It is true that the appearance of their x-ray films would suggest that distention of the remaining lung is a factor (see Fig. 4 for a representative case). But the pulmonary function studies did not demonstrate evidence of pulmonary distention in any patient. Surprisingly enough, the one in whom the studies came closest to indicating pulmonary distention was the only patient who had had a postresection thoracoplasty. His roentgenogram (Fig. 5) showed the mediastinum to be pretty much in the midline. There was only 1 patient who showed a significant degree of pulmonary emphysema (Fig. 6). But since none of the others did, we feel that it is reasonable to assume that his emphysema was a concomitant, rather than a

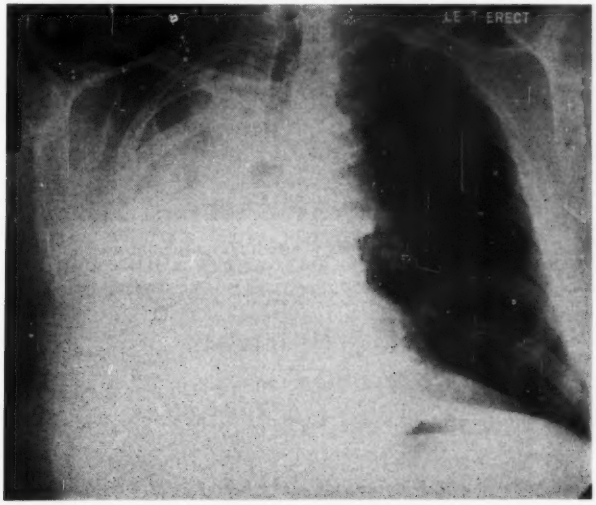

Fig. 4.—H. H.—13 years after right pneumonectomy.

consequential, complication of the resection. Evidence of pulmonary fibrosis was, on the other hand, apparent in all of the patients studied, and does therefore appear to be a related complication. Of equal importance was the finding in all cases of evidence of the presence of an increased resistance in the pulmonary vascular bed. It cannot be said whether this increased resistance is due to a loss of expansibility produced by perivascular fibrosis, or to pulmonary arteriosclerosis resulting from the long-continued increased blood flow. It may, of course, be due to both factors. Although we did not perform right heart catheterization as part of the pulmonary function studies, it is assumed from the data collected that pulmonary hypertension was present. This would be in keeping with reports published by Adams and his co-workers,[9] who found evidence of pulmonary hypertension and cor pulmonale in their post-pneumonectomy patients.

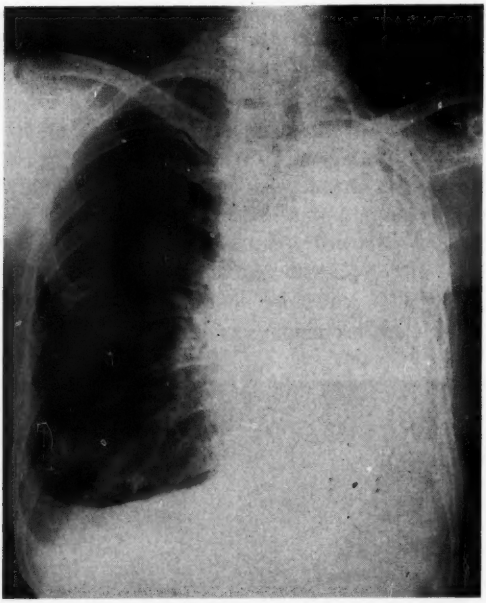

Fig. 5.—J. C.—12 years after left pneumonectomy.

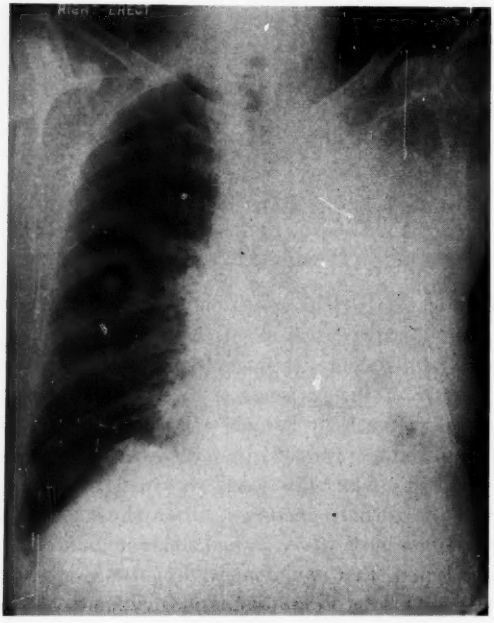

Fig. 6.—F. O. H.—16 years after left pneumonectomy.

And what has caused pulmonary fibrosis in these patients? Is it related to pulmonary arteriosclerosis, or is it due to the increased flow of air in and out of the remaining lung for a long period of time? Certainly, additional studies are needed before we can answer any of these questions. It would appear to be worth while in the future to include pulmonary function determinations in the presurgical work-up of more patients. Either then, or at the time of surgery, pulmonary arterial pressures should be recorded for future reference. Pulmonary function studies should then be repeated in those who survive resection for 1 year, and at intervals of 1 or 2 years thereafter. Information obtained from such studies might very well help us to prolong the lives of those who have had successful pulmonary resection for carcinoma. In the meantime, treatment of our long-term survivors should be directed toward relieving their bronchospasm and reducing the daily strain upon the right heart by limiting their physical activity.

SUMMARY

Thoracotomy was performed because of primary carcinoma of the lung on 513 private patients between July 1, 1942, and Jan. 1, 1959. The surgical mortality rate was 2.9 per cent. In 229 cases, exploration only was carried out, with a mortality of 0.4 per cent. In the remaining 284 patients, lobectomy was performed in 87 with one death (mortality of 1.1 per cent), and pneumonectomy in 197 with thirteen deaths (mortality 6.6 per cent), for an over-all resection mortality rate of a little under 5 per cent. Three of the 13 deaths following pneumonectomy were in patients over 70 years of age. An increased use in the commun[illegible] of routine chest x-ray examinations during the last one half of the period of study has resulted in bringing in more people with peripheral, asymptomatic lesions, and in an increase in the percentage of adenocarcinoma in the resected specimen from 12.8 to 18.1 per cent. Probably for the same reason, there has been an increase in the proportion of females from 12.2 to 13.9 per cent. Twenty per cent of 150 patients having pneumonectomy survived for 3 years or more. Of 52 patients having lobectomy, 51.9 per cent survived for 3 years or more. Especially interesting to us have been 63 patients who had pneumonectomy 10 or more years ago. Of the 33 who survived 1 year, 15 died later of carcinoma, but 7 died of causes other than carcinoma, and the majority of these deaths was attributed to cardiac failure. This fact prompted us to study the cardiopulmonary function of the 11 survivors. Of the 11, we were able to do complete pulmonary function studies on 9. Although all but 1 of these were living satisfactory lives and either working or reasonably active, they all suffered from some degree of cardiopulmonary disability. The function studies indicated that, except for 1 case, pulmonary emphysema was not a significant problem; nor was distention of the remaining lung. The abnormalities which appeared to us to be responsible for their disability are pulmonary fibrosis, bronchospasm, and increased resistance of the pulmonary vascular bed. Although not proved by cardiac catheterization, pulmonary hypertension was inferred to exist. Right heart

disease appears to be the greatest problem faced by these long-term survivors of pneumonectomy. Additional studies directed toward determining causative factors are needed.

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LUNG PERFUSION WITH CHEMOTHERAPEUTIC AGENTS

Howard Pierpont, M.D., and Brian Blades, M.D., Washington, D. C.

THE EXPERIENCE gained in the application of mechanical extracorporeal circulation methods in cardiac bypass and in hemodialysis lends itself well to administering carcinolytic agents. The perfusion of an organ isolated from its arterial and venous supply has the immediate appeal of restricting a toxic agent to that organ.

Chemotherapy in treating cancer is a relatively new and promising field. It has been limited by unfortunate side effects particularly seen in hemopoietic depression and gastrointestinal disorder. In general, these agents have a short life as far as activity once mixed with whole blood. Therefore, long perfusion periods are not necessary to deliver a maximum dose of any agent or agents.

With these principles in mind, a safe and simple technique for perfusing the lung in dogs has been developed. Such a method permits the maximum tolerated dose of a given agent to be determined for the lung tissue. It also furnishes information as to the maximum dose of the agent in terms of function of survival, once the contralateral lung is removed.

It is hoped that such a method will prove useful as a form of bioassay of existing chemotherapeutic agents as well as those to be developed in the future.

Clinical application of this technique is under study. If it proves to be practical, the surgeon may have a method of palliation to offer those patients with inoperable lung cancer at the time of thoracotomy.

METHOD

Operation.—Dogs weighing forty pounds or more were anesthetized with intravenous Pentothal sodium and placed on intermittent positive pressure oxygen flow administered through an endotracheal tube with inflated cuff. A left fifth rib thoracotomy was performed and the left pulmonary artery and veins were exposed by incising the pericardium at its reflection over these vessels. The left pulmonary artery was mobilized and occluded by a double loop of umbilical tape just lateral to the conus. The left half of the atrium was then freed from the trachea and posterior mediastinal space to allow a Satinsky clamp to occlude the atrial chamber, lateral to the anterior descending coronary vessels.

The George Washington University, School of Medicine, Washington, D. C.

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After a second umbilical tape was fixed around the pulmonary veins to control blood loss, a 15 mm. incision was made in the lateral wall of the auricle just above the origin of the inferior pulmonary vein. Using No. 1 braided silk suture on an atraumatic round needle, a purse-string ligature was placed in the margin of the cardiotomy. This suture served to fix a Lucite button in the wall of the atrial chamber. The button (Fig. 1) was designed in the laboratory for the experiment and, once in this position, has never obstructed the outflow of venous blood to the perfusion circuit.

The pulmonary artery was then cannulated by a flanged, curved polyethylene tubing (PE 980).

No attempt was made to occlude the bronchial circulation.

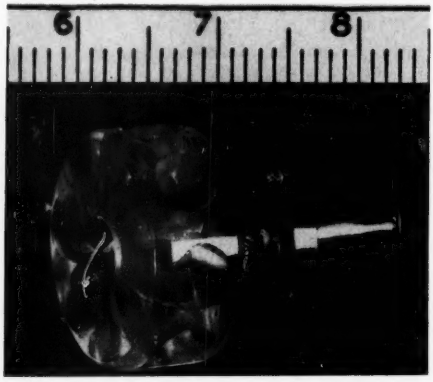

Fig. 1.—Enlargement of Lucite "button" designed to collect blood from the left half of the atrial chamber. The grooved central portion anchors the "purse-string" sutured cardiotomy in position. Ratio of collecting inlet diameter to outlet diameter is 9 to 1. The tip of the "button" fits a 0.5 cm. I.D. Tygon tubing.

The outflow and inflow cannulae were allowed to fill with blood from the isolated left lung. Clotting was prevented by administering intravenous heparin to the animal prior to the cannulation. A dose of 0.8 mg. per kilogram body weight was used. These cannulae were then connected to the perfusion apparatus.

After perfusion, the cardiotomy was closed with a running suture of 4-0 silk and the Satinsky clamp was released after precautions were taken to prevent air emboli. The pulmonary artery incision was closed with 5-0 silk in a transverse fashion which prevented stenosis. The chest wall was then closed over a water-seal drain.

Perfusion Technique.—The perfusion apparatus consisted of a Sigmamotor pump, a 500 ml. siliconized collecting bottle, and Tygon tubing connections to the cannulae. Just proximal to the pumping point, a right angle connection was attached to a 50 ml. titrating burette which was equipped with a stopcock and Murphy drip chamber (Fig. 2).

The entire perfusion circuit was sealed so that the pump not only propelled blood through the pulmonary artery but created a balanced negative pressure

in the reservoir which aided the outflow of blood from the pulmonary veins. Two hundred and fifty milliliters of whole blood, anticoagulated with heparin, was used to prime the perfusion circuit. The priming level remained constant throughout the perfusion period.

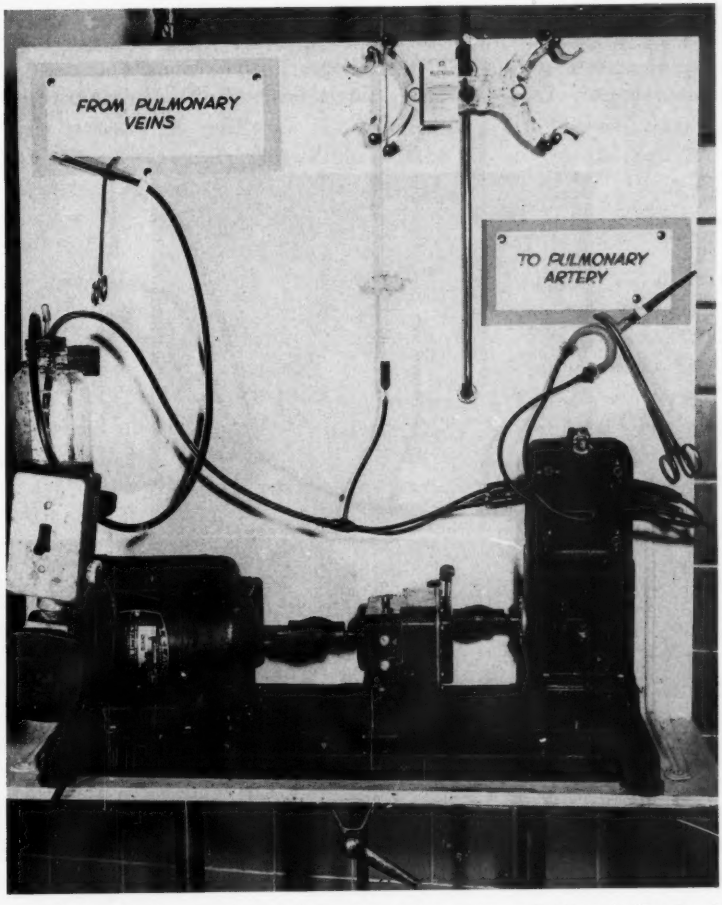

Fig. 2.—Vertical adjusting rack mounting Sigmamotor pump, collecting bottle, burette for chemotherapeutic agent, and disposable Tygon connecting tubes for perfusing the left lung.

The titrating burette contained a freshly prepared solution of nitrogen mustard* in isotonic saline which was added drop by drop to the circuit after the perfusion was begun.

The flow rate of the recirculating blood was maintained at 175 ml. per minute. The nitrogen mustard solution was added over the first 2 to 3 minutes of a 15-minute perfusion time.

*Mustargen (methyl-bis-[beta-chlorethyl]-amine hydrochloride), Merck & Co., Inc., Philadelphia, Pa.

No attempt was made to wash out the perfusion blood from the pulmonary vascular bed in this series. This was based on the observation made by Creech, Krementz, Ryan, and Winblad[1] that nitrogen mustard becomes inactive or fixed in whole blood in approximately 8 minutes.

Dose of Nitrogen Mustard.—In the first 10 perfusions, random dosages of 0.2 to 0.7 mg. per kilogram of total body weight were used. There were no survivals in dosages above 0.4 mg. per kilogram. The remaining twenty-three perfusions therefore were made using this amount of nitrogen mustard.

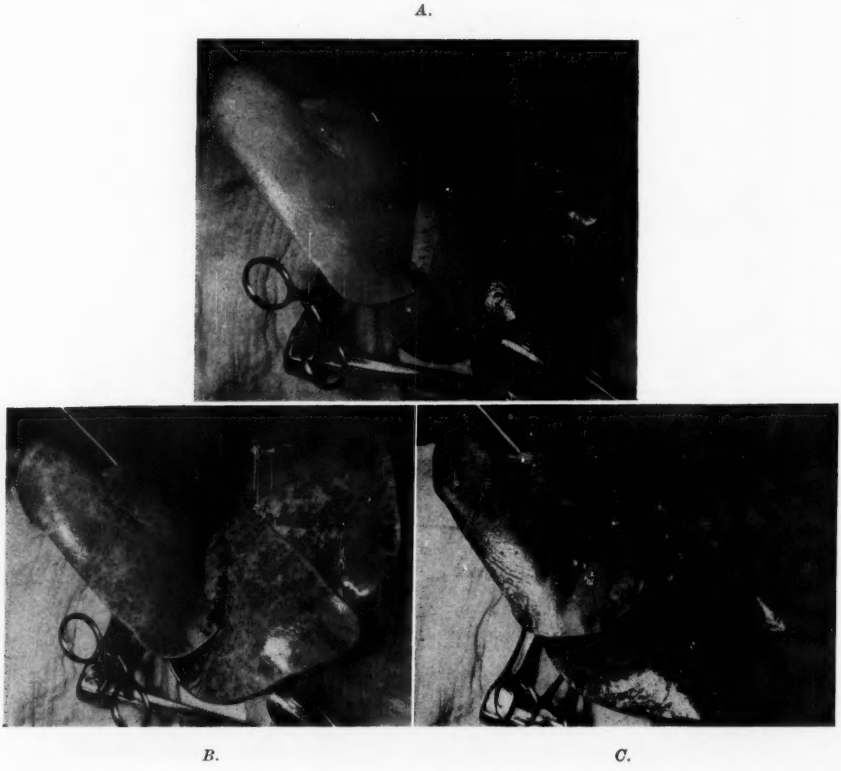

Fig. 3.—Illustration of the dispersion of methylene blue dye in the whole blood perfusion of animal's left lung. *A*, Before injection of dye. *B*, Three minutes after instillation of dye in perfusate. *C*, Five minutes after instillation of dye in perfusate.

The Perfusate.—Whole blood was selected to minimize pulmonary edema. Saline and plasma were tried but found to be unsatisfactory because a crepitant wet lung developed rapidly, early in the perfusion.

Radioactive chrome-tagged red blood cells were added to the perfusate during initial studies to determine the escape of the perfusate to the host circulation. In five separate perfusions, no trace of the radioactive element could be found in peripheral blood samples taken at the completion of the experiments.

The rapid and even distribution of the perfusate throughout the lung tissue was confirmed by infusion of methylene blue dye. Within 5 seconds after the column of dye was seen to enter the pulmonary artery, all divisions of each lobe were seen to be uniformly stained with the dye (Fig. 3).

The rate of flow of the perfusate was another important factor in preventing pulmonary edema. Initially, rates of 300 ml. per minute were found to cause a high incidence of edema, congestion, and bronchial effusion. These complications were controlled by reducing the rate of flow to 175 ml. per minute.

Oxygenation of the perfusate was supplied by the tracheobronchial oxygen flow from the resuscitator. This eliminated the need for an oxygenator in the perfusion circuit.

RESULTS

Results of this experiment fall into three categories: (1) survival of the animal using the maximum tolerated dose of nitrogen mustard, (2) evidence of systemic toxic effect based on periodic red blood cell and white blood cell counts, and (3) histologic evaluation of the perfused lung tissue at intervals following perfusion.

In a series of 23 consecutive animals, all perfused with 0.4 mg. nitrogen mustard per kilogram total body weight at 175 ml. per minute, there were 10 survivors. Of the remaining 13 animals, 2 died of acute pulmonary edema, one of cardiac arrest, one from pneumothorax, one from hemorrhage at the cardiotomy site, 3 from pneumonitis, and 5 from distemper.

The 10 validated survivors are all well and the effect of the nitrogen mustard on the peripheral blood count is tabulated in Figs. 4 and 5. Three of the 10 animals had an appreciable depression of the red blood cells and only one showed a leukopenia. It is possible that distemper again played a role as the anemia was not uniform throughout the group.

Histologic study of perfused lung tissue is not yet complete but in this preliminary series there are specimens covering 1 to 35 days following the operation. In general, the histologic changes are similar to acute pneumonitis with resolution in that there is a sequence of vascular effusion and hemorrhage, cell necrosis, monocytic infiltration, and later fibrosis.

DISCUSSION

The ability to perfuse one lung without interrupting the general circulation is a distinct advantage of this experiment. In 1953, we found the dog's lung can be isolated from its pulmonary and bronchial blood supply for 30 minutes and survive contralateral pneumonectomy.[2] The present experiment indicates one lung can be isolated from the host and perfused with a dose of nitrogen mustard, 175 to 200 times that which can be given intravenously. The capacity of lung tissue to withstand such a chemical insult offers a new technique for administering chemotherapeutic agents. This method is an improvement on the intravenous and intra-arterial routes in that a higher concentration of the agent may be fixed in the lung tissue without disturbing the bone marrow.

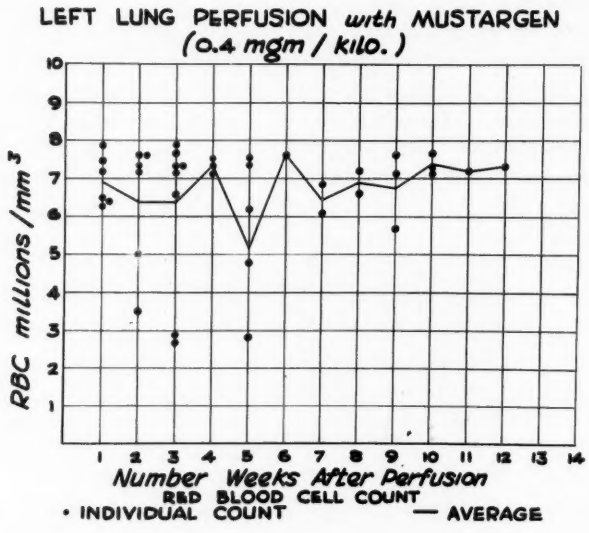
LEFT LUNG PERFUSION with MUSTARGEN
(0.4 mgm / kilo.)
RBC millions / mm³
1 2 3 4 5 6 7 8 9 10
1 2 3 4 5 6 7 8 9 10 11 12 13 14
Number Weeks After Perfusion
RED BLOOD CELL COUNT
• INDIVIDUAL COUNT
— AVERAGE

Fig. 4.

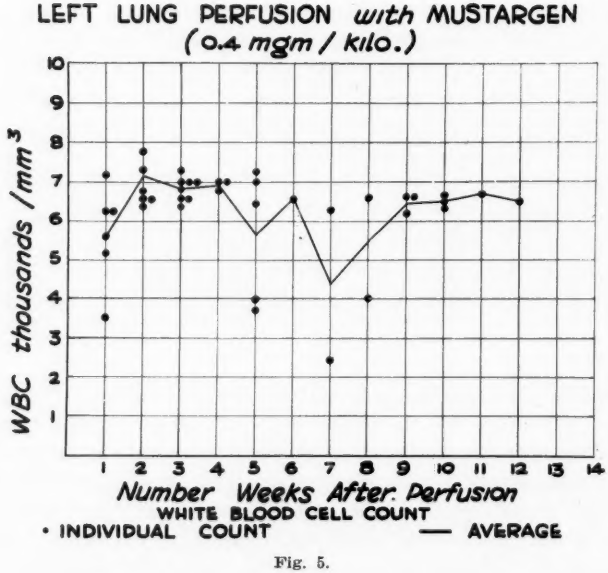
LEFT LUNG PERFUSION with MUSTARGEN
(0.4 mgm / kilo.)
WBC thousands / mm³
1 2 3 4 5 6 7 8 9 10
1 2 3 4 5 6 7 8 9 10 11 12 13 14
Number Weeks After Perfusion
WHITE BLOOD CELL COUNT
• INDIVIDUAL COUNT
— AVERAGE

Fig. 5.

The relative increase in the maximum tolerated dosage of nitrogen mustard given by isolated perfusion over that given intravenously is based on the relative weight of the left lung compared to the total body weight. In dogs, this ratio is 1 to 195.

The failure of chrome-tagged red blood cells to escape from the perfusion circuit to the general circulation is surprising. This tends to support the theory that bronchial veins drain into the pulmonary venous system. The escape of noncellular elements of the perfusate via the lymphatics is the subject of a later report.

The effects of this type of administration of chemotherapeutic agents on lung tumor await clinical application. Palliation is all that can be hoped for using existing compounds. However, it is to be expected that a regression and even reduction in size of the lesion may make certain tumors resectable at a later date.[3]

The experiments described supply a laboratory preparation for study of the effect of specific agents on lung tissue and furnish a method of deriving basic information for each agent before clinical application.

SUMMARY

1. An experimental method for the isolated perfusion of one lung with high concentrations of chemotherapeutic agents for the palliation of cancer is presented.

2. Concentrations of nitrogen mustard, two hundred times that tolerated by intravenous injection, were given without the toxic effect of bone marrow depression.

3. Radioactive chrome-tagged red blood cells do not escape the perfusion circuit in this technique.

4. Nitrogen mustard, 0.4 mg. per kilogram of total body weight, dissolved in isotonic saline is the maximum tolerated dose in perfusion of the dog's lung.

5. The method provides a laboratory bioassay technique for existing chemotherapeutic agents, as well as experimental ones, to be used in palliation of lung cancer.

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SEVERE CRUSHING INJURIES OF THE CHEST

A Simple Method of Stabilization

L. H. Strug, M.D., B. Glass, M.D., W. Leon, M.D., and M. Salatich, M.D., New Orleans, La.

SEVERE crushing injuries of the chest have become more important to physicians in recent years because of the increasing incidence of major thoracic trauma attendant with high speed vehicular accidents. The high mortality and morbidity associated with such injuries emphasize the necessity for the general practitioner and the general surgeon to be familiar with the management of such injuries. Proctor and London[1] report that of 344,000 injuries treated during the 9-year period 1944-1952, there were only 8 chest injuries with paradoxical respiration requiring surgical treatment. Rives and Kahle,[2] reporting in 1939 on chest injuries following automobile accidents, found that of 16,000 accident patients seen in New Orleans Charity Hospital accident room in a 6-month period, only 24 required hospitalization. On the other hand, Chandler[3] states that chest injuries comprise 25 per cent of all traffic deaths. Chest injuries of the crushing type, such as rib fractures associated with pleural or lung complications, are generally conceded to carry a 10 to 20 per cent mortality rate.[4] As automobiles continue to increase in horsepower, it appears reasonable to conclude that severe crushing injuries of the chest will continue to increase in frequency.

In the 3-year period 1955-1958, there were 154 cases of nonpenetrating chest trauma of sufficient severity to require hospitalization that were admitted to the Louisiana State University Thoracic Surgical service of New Orleans Charity Hospital. It would be impractical to ascertain exactly how many victims of nonpenetrating chest trauma were treated in the accident room without hospitalization, but a conservative estimate would place it at approximately twice the number of those admitted. This estimate is based both on observations by the authors and by the ratio of 3 to 1 found by previous investigators from this institution over a shorter period of time.[2] However, it is readily apparent without resort to statistical analysis that severe thoracic trauma is on the increase.

Of the 154 patients admitted, there were 39 cases involving either a flail chest, or a fractured sternum. There were 8 fractures of the sternum with

From the Department of Surgery, Division of Thoracic Surgery, Louisiana State University School of Medicine, and Charity Hospital, New Orleans, La.

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hemothorax, one case of fractured sternum with hemopneumothorax, and one case of fractured sternum with injury to the heart. There were 10 cases of flail chest with paradoxical motion, but without intrathoracic injury, 2 cases of flail chest with hemothorax, 2 with pneumothorax, 7 with hemopneumothorax, 1 with diaphragmatic rupture, and 2 with cardiac injury. Of these 39 patients, 15 required external traction of some sort to stabilize the chest wall, 9 required sandbags for stabilization, and 2 required open thoracotomy. The remaining 13 required no type of stabilization, either because of the location of the unstable area or because of minimal degree of paradoxical respiration. All patients with hemothorax, pneumothorax, or hemopneumothorax were treated with either single or multiple thoracenteses, or with closed intercostal catheter drainage into a water trap, or with appropriate negative pressure suction. Of the 39 cases of severe crushing injuries of the chest, there was one death. Autopsy revealed a severe cerebral laceration in addition to the chest injury.

Intelligent management of crushing injuries of the chest requires a thorough knowledge of the normal physiology of respiration and of the disturbances of the physiology brought about by the particular type of injury under consideration.[5, 6] The chest wall is "stove in" so that it loses its rigidity and becomes a flail chest. If the crushing force is from the lateral direction, the pathology of the injury usually consists of fractures in at least two places of multiple adjacent ribs on the side involved. This results in a central portion of the chest wall which is floating. This floating section moves in and out with respiration in a reverse or paradoxical direction to the rest of the chest wall. This type of respiration markedly decreases the efficiency of ventilation and is usually accompanied by severe pain that renders the coughing mechanism ineffectual. More often than not there is associated a hemothorax of considerable degree, together with marked contusion and often laceration of the lung, with extravasation of blood, edema, and retention of secretions[6] in the tracheobronchial tree.

If the blow is directly over the sternum, as frequently occurs in steering wheel injuries, fractures of the sternum may occur. This type of injury is most frequently associated with bilateral costochondral fractures, usually from the first rib down, resulting in an extreme flailing of the anterior portion of the chest wall.

Recognizing the above concept of the disturbed physiology resulting from the flail chest with paradoxical motion, it becomes evident that large areas of flailing must be stabilized in order to restore as closely as possible the normal physiology. The surprising thing to the physician observing this phenomenon for the first time is the amount of respiratory distress, anxiety, pain, and shock which can and does result on occasions from apparently small areas of paradoxical chest movement. One is equally impressed by the marked improvement in respiration, lessening of anxiety and

pain, mental clearing, and the return of normal cardiac action and blood pressure following some form of external fixation of the flailing segment of chest wall.

As can be seen from the treatment employed in our cases, external fixation may be accomplished utilizing a variety of methods. A perusal of the literature reveals many proposed methods of fixation, ranging from simple manual pressure over the flailing area as an emergency method, adhesive strapping of the involved side, sandbags to compress and stabilize the involved weakened segment, rolling the patient onto the flailing area,[7] traction exerted on stainless steel pins passed through the pectoral muscles over the crushed area,[8] towel clips or wires passed around the ribs or sternum in the involved area,[9] cup hooks used for sternal traction,[10] internal fixation of fractured ribs by intramedullary pins and/or wiring of fragments,[11] to application of metal plates to the fragments of fractured sternum.[12]

We have utilized manual pressure as well as turning the patient onto the involved side so as to splint the thoracic wall by the patient's own weight. This is particularly applicable in posterior fractures. Here we have the heavy thoracic muscles which act as a splint to prevent paradoxical motion, or minimize its extent.

We have used sandbags applied over a lateral or anterolateral area of flailing to compress and stabilize the area with good results. It must be admitted that with sandbags the area of compression causes a decreased volume of the involved hemithorax and a consequent decrease in the exchange of air on that side. However, the vicious and dangerous cycle of paradoxical motion, mediastinal flutter, and "pendlehuft" motion is eliminated and more than compensates for the slight decrease in total lung volume on the involved side. Adhesive strapping is mentioned only to condemn it as anything other than an emergency measure for short periods of time in order to transport a patient from the scene of an accident to a hospital. Adhesive strapping, to be effective, must be carried completely around the involved side and past the midline onto the normal side both anteriorly and posteriorly. This compresses and restricts respiratory motion over a large area of normal chest wall as well as over the flailing portion. The use of rib belts, girdles, or other encircling splints is also condemned. Traction exerted on Steinman pins passed through the pectoral muscles is particularly useful in large anterior flailing chest wall segments.[8] We have used this method on occasion in addition to a method of sternal traction which will be described later. This method of traction in anterolateral segments is preferred where sandbagging seems inadequate. Towel clips applied to the ribs or sternum have been used in the past,[9] but they have been used in the present series only as emergency measures until some other method of traction could be instituted. Our chief objection to towel clips and to wires lies in the fact that both clips and wire have a tendency to cut through the tissues, even through bone and cartilage, and have at times come completely loose in as little as 48 hours, which is considered inadequate time for stabilization of a flailing chest. We

have had no experience with intramedullary pins or metal plates. Thoracotomy, with its attendant added risk, is reserved for those patients having either actual loss of chest wall requiring surgery for adequate closure of a penetrating wound, or intrathoracic complications which make immediate thoracotomy mandatory. In these cases we have resorted to wiring of the fractured rib fragments with very satisfactory results.[11]

For the last 10 years, we have utilized a simple method of sternal traction which we think offers many advantages, particularly in fractures of the sternum with flailing and in fractures over large segments of the costochondral area. In 1949, two of us (L. S. and M. S.) after observing the use of external fixation of the mandible by the maxillofacial surgeons adapted this principle to external fixation of sternal fractures and extended its use to sternal traction for such fractures and for costochondral fractures.

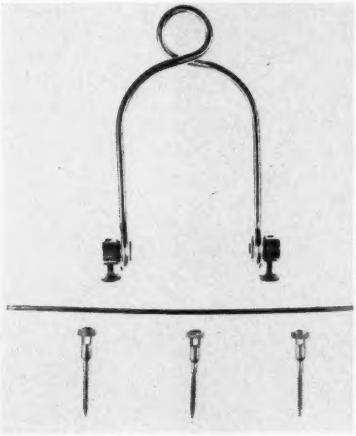

Fig. 1.

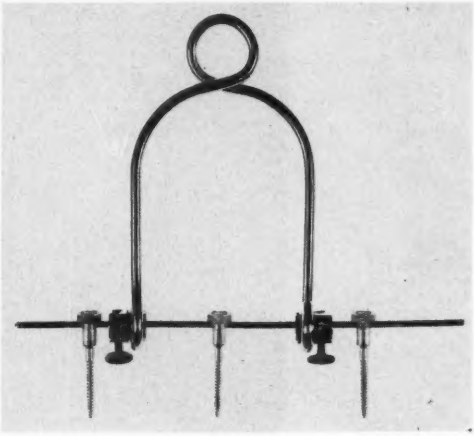

Fig. 2.

Fig. 1.—Bigelow screws—metal bar and traction spreader unassembled.
Fig. 2.—Bigelow screws—metal bar and traction spreader assembled.

The equipment used consists of Bigelow screws,* a pliable aluminum bar which fits into the slotted screws, a traction spreader (Fig. 1), a drill and bit, screwdriver, Balkan frame and weights. Everything except the Bigelow screws and bar are standard hospital equipment. The screws and bars are standard equipment on a maxillofacial service and are relatively inexpensive. On our service, a complete set is always kept sterile in the emergency accident room. Illustrated in Fig. 2, are the Bigelow screws, bar, and spreader. The screws are self-threading and each is provided with a slotted head and a retaining nut which screws over the slotted head.

The technique of insertion of the screws into the sternum is simple, requires only small amounts of local anesthesia, and is relatively bloodless.

*Bigelow screws manufactured by Austenal Laboratories, Inc., Surgical Division, 224 East 39th Street, New York 16, N. Y.

Utilizing local anesthesia, skin wheals are raised at the proposed sites of insertion of the screws. Usually two screws are inserted in the midline above and two below the site of a sternal fracture. On occasion the fragments may be so comminuted that only one screw may be placed in a fragment. On rare occasions, because of multiple fragments, more than four screws must be used (Fig. 3). Anesthesia is infiltrated down to the periosteum of the sternum at the proposed sites of screw insertion. At each site, a small stab wound is made down to the sternum and a hand drill with the Crutchfield drill point* is used to perforate the anterior cortex of the sternum. The drill point is guarded so effectively that there is minimal danger of penetrating

Fig. 3.—Blown up view of traction apparatus applied to sternum.

any further than the anterior cortex. The Bigelow screw is then inserted into each hole so formed and screwed into place. When the posterior cortex of the sternum is encountered there is a very definite sensation of increased resistance, and there is a lessening of this resistance when the posterior cortex is penetrated by the screw. Thus, with a modicum of caution and tactile sense, the operator may blindly insert the screw far enough to ensure a firm, strong fit, yet not so far as to impinge upon the heart or great vessels. If desired, a lateral x-ray of the sternum may be utilized to check the depth of the screws. However, we do not find this necessary. After insertion of all screws, the slots in the heads of the screws are aligned. The malleable

*Crutchfield drill point manufactured by Codman and Shurtleff, Inc., 104 Brookline Avenue, Boston 15, Mass.

bar is fitted into the slots, bending the bar as necessary to fit the slots. The retaining nuts are screwed onto the head of the screws so as to retain the bar in place. Not infrequently, in the case of sternal fractures with paradoxical motion, no further fixation is needed. However, should costochondral or parasternal chondral fractures exist, traction will be necessary. This is accomplished by placing the patient in an orthopedic bed and running a rope from a clevis placed on the malleable bar up to an overhead pulley, thence to a pulley at the foot of the bed and adding from three to five pounds of weight as may be needed to stabilize the chest. In the case of parasternal and costochondral fractures, the traction on the sternum is extended through the soft tissues of the anterior chest wall to the site of the fracture. On

Fig. 4.—Double bar traction to sternum with bilateral costochondral rib fractures and with weights attached.

rare occasions the segment of flailing has been so large that lateral muscle traction of the Hudson type[8] has also been necessary. A double bar with Bigelow screws may be necessary on rare occasion (Figs. 4 and 5).

We feel that the advantage of simplicity of performance of this procedure and the avoidance of a general anesthesia should definitely be considered as opposed to thoracotomy with internal fixation. We consider that the advantages of this procedure, as opposed to wires or towel clips, lie not only in simplicity and avoidance of possible pneumothorax, but primarily in

the fact that properly placed Bigelow screws will remain in place much longer without cutting through the tissues. We have frequently utilized up to 12 pounds of traction for 2 weeks. This period allows sufficient healing to occur so that paradoxical motion is eliminated when the traction is released. Subsequently the screws and external fixation bar may be left in place for another 2 weeks to ensure firm union of a sternal fracture. Occasionally there may be a semipurulent drainage from the screw holes for a few days, but we have had no cases of osteomyelitis. We believe that the extra fixation afforded by the malleable bar fitting into the screw heads provides a definite advantage over the modified cup hooks described by Rook[10] and utilized by Proctor and London.[1]

In addition to the use of some form of fixation of the chest wall, attention must be centered on the maintenance of an adequate airway. This has been emphasized by others.[13, 14] We frequently utilize nasotracheal suction and/or bronchoscopy. It has been our experience that the vast majority of

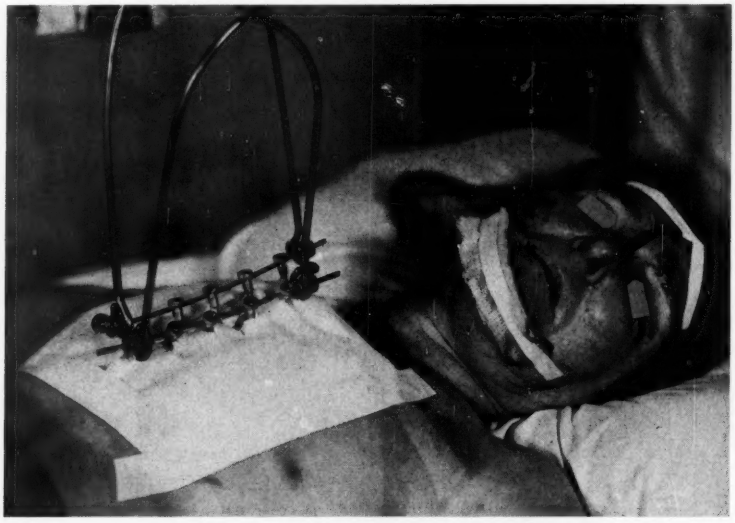

Fig. 5.—Blown up view of double bar traction to sternum in bilateral costochondral fracture with severe flailing of chest.

these patients cannot be kept free of accumulated secretions without repeated nasotracheal aspiration. In addition to predisposing to laryngeal edema and further respiratory embarrassment, nasotracheal aspiration requires the constant attendance of a skilled and energetic nurse or physician. The same, with even more emphasis on the skill and energy of the physician, is true of repeated bronchoscopy. Still another shortcoming of these modes of treatment lies in the fact that they do nothing to cut down on the dead air space. The addition of a tracheotomy has been shown to decrease the dead air space by 75 to 150 c.c.[13] Aspiration of secretions may then be carried out by relatively unskilled help, there is less resistance to air movement, hence less

paradox, and oxygen consumption is decreased because of decreased muscular exertion needed to breathe.[14] Therefore, if the patient is having significant flailing or any signs of respiratory distress or hypoxia, we routinely resort to early tracheotomy.

The treatment of shock should follow the usual lines of blood replacement, nasal or tracheal oxygen, relief of pain, etc. Frequently, the treatment of the primary injury of a flail chest will alleviate the clinical signs of shock without blood or plasma expanders having to be used.

The treatment of intrathoracic complications, such as hemothorax and/or pneumothorax, must, of course, be handled intelligently in these cases. If significant hemothorax or tension pneumothorax exists, we prefer to utilize intercostal catheter and water trap drainage with appropriate suction.[5] However, in cases of hemothorax and/or pneumothorax of lesser degree we often use either single or multiple thoracenteses followed by frequent, portable x-ray studies. The details of treatment of complications are not the province of this paper. Suffice it to say that we believe the best method of sealing off a leak of air or blood from the lung lies in rapid re-expansion of the lung, approximating visceral to parietal pleura. Whether this is accomplished by needle aspiration or intercostal drainage is relatively unimportant. Thus, we individualize the treatment of these complications.

Necessarily, an injury of sufficient severity to crush a chest will usually produce multiple injuries. The appropriate treatment of orthopedic, abdominal, neurological, genitourinary, and maxillofacial injuries must be carried out concomitantly. We would agree with Daughtry[6] that joint cooperation and consultation between various specialists should be utilized. However, in general, the most rapid killer in the group of multiple injuries will usually be the chest injury unless it is handled intelligently and promptly. The general surgeon with a good orientation toward thoracic physiology, or in consultation with the thoracic surgeon, should be able to treat adequately these severe and dangerous injuries.

SUMMARY

Severe crushing injuries of the thorax are definitely on the increase. As a result, flail chest with paradoxical motion is seen more often.

Disturbances in respiratory physiology occur and it is mandatory that these be remedied immediately. In order to do this, the area of flailing must be stabilized.

Although many methods of stabilization have been reported, and are currently used, none are completely satisfactory.

A simple method of sternal fixation and traction utilizing the Bigelow screws and bar is presented, and our experiences with this method are related.

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MIDDLE LOBECTOMY FOR TUBERCULOSIS IN CHILDHOOD

David E. Thomas, Lieutenant Colonel, MC, USA, Phoenixville, Pa., and Dean Winn, Major, MC, USA,** Denver, Colo.*

REVIEW OF THE LITERATURE

THE middle lobe syndrome has received a great deal of attention since Hampton and King[1] described the roentgenographic changes consistent with atelectasis of the middle lobe and demonstrated that what was called interlobar empyema, pleural fluid, or thickened interlobar septa was generally an airless middle lobe. Kent,[2] in 1942, discussed 10 cases of atelectasis of various lobes associated with tuberculous enlarged hilar nodes in childhood. He showed that the homogeneous density seen in x-ray studies did not represent tuberculous pneumonia. Seven of his 10 patients became bronchiectatic in the area of involvement after 7 months or more of chronic bronchial occlusion. He felt that the prognosis was favorable without operation. Jones, Peck, and Willis[3] also demonstrated bronchiectasis, or no filling on bronchogram, in 70 per cent of a sizable series of cases of primary tuberculosis with intrinsic or extrinsic bronchial obstruction examined 1 year after the original diagnosis.

The pathogenesis of endobronchial tuberculosis in children is presented in a very thorough and complete article by Daly, Brown, Lincoln, and Wilking.[4] They prove bronchoscopically that endobronchial tuberculosis is common in children with tuberculosis, particularly in younger children, and that the middle lobe is most frequently involved. Bronchoscopy revealed compression or fixation by nodes, sometimes obstruction by mucosal inflammation, granuloma, caseous material extruded from a ruptured node, or a fibrous stricture. Progression from one lesion to another was observed. Late manifestations of bronchiectasis, persistent roentgenographic shadows, productive cough, râles, and recurrent lung infection are described.

Graham, Burford, and Mayer,[5] in 1948, reported 12 resections for the middle lobe syndrome, all in adults, all with good results, and none for tuberculosis. They emphasized the vicious circle of obstruction causing infection which resulted in more lymphadenitis. Shaw and Paulson,[6] in 1949, stated that a collapsed middle lobe may or may not be associated with enlarged or calcified lymph nodes, may be due to mucosal inflammation, foreign body, or any other form of obstruction. Middle lobectomy was recommended and chronic pneumonitis, atelectasis, fibrosis, and bronchiectasis were described in the resected

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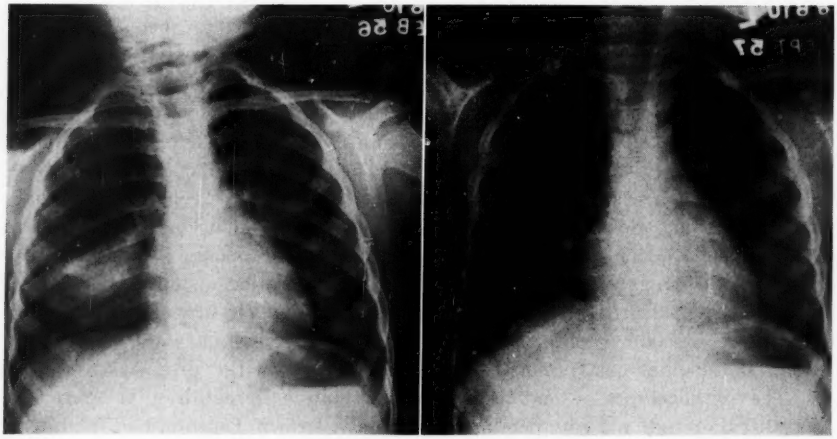
*Valley Forge Army Hospital, Phoenixville, Pa.
**Fitzsimons Army Hospital, Denver Colo.

specimens. Resection is also advised for the middle lobe syndrome by Harper, Condon, and Wierman,[7] who feel there is no definite syndrome associated with middle lobe collapse. Tuberculosis was found in 3 of 26 specimens, the remaining ones showing chronic pulmonary suppuration and bronchiectasis with fibrosis.

There is no unanimity of opinion concerning the treatment of choice in middle lobe atelectasis due to childhood tuberculosis. Perusal of the case reports will show that our 3 patients were discussed at great length repeatedly before undergoing thoracotomy and, in each case, surgery was performed although several physicians did not concur. In view of the controversy these little patients stimulated, an examination of their cases seems worth while.



A. *B.*

Fig. 1.—Case 2. *A*, Before surgery. *B*, One year after surgery.

CASE REPORTS

CASE 1.—This 4-year-old patient contracted tuberculosis in Germany at the age of 15 months. He received streptomycin and para-aminosalicylic acid (SM-PAS) therapy for 14 months. Two years later, due to roentgenographic changes and a palpable lymph node in his neck, he was admitted to Fitzsimons Army Hospital. At this time the middle lobe was atelectatic. The child was asymptomatic.

Physical examination revealed an occasional wheeze in the right middle lobe area, bacteriologic examinations were negative, bronchograms demonstrated no filling of the middle lobe, and bronchoscopy showed granulation tissue obstructing the right middle lobe bronchus. Repeat bronchoscopy 5 months later showed a clean, narrowed right middle lobe orifice.

The patient was presented at staff conference four times in 5 months, and there was wide disagreement regarding the need for surgical intervention. A middle lobectomy was performed, since this was the desire of the majority. A nodose lesion was also locally excised from the anterior basal segment. The postoperative course was benign. At surgery, the middle lobe orifice was noted to be markedly stenotic.

Pathologic examination revealed bronchiectasis, grossly, and pulmonary fibrosis, microscopically, in the middle lobe. Tubercle bacilli were identified in the lesion from the anterior basal segment.

X-ray studies 3 years later revealed only minimal pleural changes at the right base. The child is well.

CASE 2.—This 3-year-old child contracted tuberculosis in Turkey when but a few months old. She arrived at Valley Forge Army Hospital 2 years later on SM-PAS therapy. She was asymptomatic. Films dated February, 1956, showed a right middle and lower lobe pneumonic infiltrate. The lower lobe lesion cleared but middle lobe opacity was still present 7 months later. Bronchograms did not reveal bronchiectasis. Gastric cultures were positive for tubercle bacilli. Bronchoscopy failed to reveal the etiology of the middle lobe atelectasis.

Lobectomy was recommended the first time this patient was presented at staff conference. Thoracotomy 8 months after admission revealed a collapsed lateral segment of the middle lobe. In the resected specimen, a fibrin plug was found occluding the bronchus. There was no evidence of extrinsic pressure or intraluminal disease. Microscopically, no evidence of tuberculosis was found in the resected specimen. The postoperative course was uncomplicated, and 1-year follow-up revealed a normal child with a normal roentgenogram of the chest.

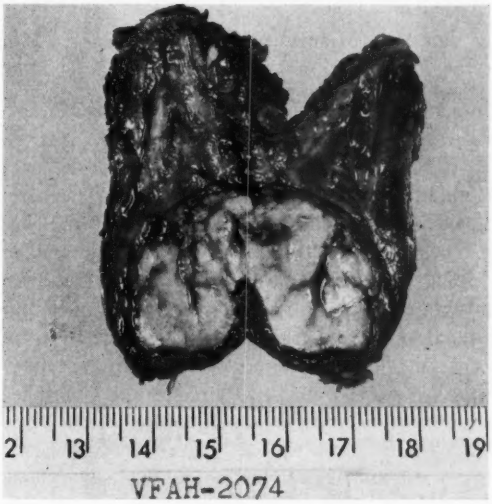


Fig. 2.—Case 3. Middle lobe opened to show caseo-cavitary lesion.

CASE 3.—This 2-year-old Japanese-American child who was born in Japan had a chest roentgenogram taken prior to circumcision. This revealed what was interpreted as a pneumonic process in the middle lobe. As PPD 1 was found to be positive, he was transferred to Valley Forge Army Hospital where one gastric culture revealed *Mycobacterium tuberculosis* resistant to isonicotinic acid hydrazide (INH) as well as being catalase negative. The child was asymptomatic. INH-PAS therapy for 6 months did not alter the roentgenographic appearance of middle lobe atelectasis.

At staff conference this child was considered repeatedly for surgery at the request of the Pediatric Service. A variety of opinions were expressed by house and consultant staff and, although lobectomy was finally authorized, no unanimity of opinion was reached. A middle lobectomy was performed after 7 months of antituberculous therapy. A large caseo-cavitary lesion with wide open bronchial communication was found. The postoperative course was benign and roentgenograms made 1 month after surgery revealed only minimal postoperative pleural changes.

DISCUSSION

It is apparent that atelectasis of the middle lobe may be an asymptomatic lesion. A proper etiologic diagnosis is not always possible prior to surgery. Had we known that a fibrin plug caused the atelectasis in Case 2, surgery would not have been performed. On the other hand, all would have recommended surgery in Case 3 had we known that there was an open cavitary lesion in the middle lobe. On the basis of probability, one would expect to find such lesions as bronchiectasis, pulmonary fibrosis, bronchostenosis, or extrinsic lymph node pressure, as reported by others.

It is well known that patients of all ages do well after middle lobectomy, since the architectural arrangement almost precludes the commonest postoperative complications, bronchopleural fistula and space problems. These young children had smooth postoperative courses, can proceed through life without demonstrable tuberculous lesions, and further tuberculous activity may have been avoided in 2 cases.

As a warning, this type of surgery is often not easy. Dense fibrosis about the hilum makes the identification and isolation of arterial branches and the middle lobe bronchus a tedious and time-consuming process.

SUMMARY

Three cases of middle lobe syndrome from primary tuberculosis in infancy and young childhood are presented. The treatment of choice is lobectomy after an adequate period of antituberculous therapy. The etiology of the atelectasis is multiple. The pathologic findings vary and are not always predictable. An uneventful postoperative course can be anticipated.

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PARTIAL RESECTION OF THE ONLY REMAINING LUNG WITH THE AID OF RESPIRATOR TREATMENT

Viking Olov Björk, M.D., Uppsala, Sweden

INDICATIONS

RESPIRATOR treatment is indicated in every case of ventilatory insufficiency regardless of its cause, if bronchial aspirations and a tracheostomy alone do not bring the pCO_2 back to a normal level.

Ventilatory insufficiency will occasionally be found in the postoperative course after any extensive intrathoracic procedure on either the lung and the chest wall, the heart, the great vessels, or the esophagus. At this stage, when the patient's own respiratory work is insufficient and accompanied by carbon dioxide retention, respirator treatment may be lifesaving.

When is then the right moment for introduction of artificial ventilation? Clinical experience and a careful observation of these patients will usually make the decision easy. If one is doubtful, an arterial puncture with determination of the pCO_2 is helpful.

Sufficient experience has now accumulated so as to make possible, in certain cases, the decision of postoperative respirator treatment even before the operation. In this event, all respiratory work is taken away from the patient in the postoperative period and an adequate ventilation is guaranteed. Furthermore, the patient is relieved of all effort needed to bring up accumulated secretion as this will be aspirated at intervals. There will be no pain as the patient can receive sufficient morphia to control it without regard to its effect on the respiratory center. Resection of part of the only remaining lung is considered a clear indication for postoperative respirator treatment. The first such patient operated upon before the introduction of the respirator in thoracic surgery died on the second day due to an aspiration pneumonia. Four subsequent patients have survived, mainly due to the respirator treatment.

TECHNIQUE

After an experience of more than 200 cases of postoperative respirator treatment, certain technical details have proved important.

The Tracheostomy.—

A transverse skin incision is preferred. It should be placed as low down on the neck as possible for cosmetic reasons. In some cases, the skin will be

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fixed to the trachea when the cannula is removed causing discomfort to the patient when swallowing. Then a plastic operation is required to free the skin from the trachea, and to interpose muscles in the midline between the skin and the trachea. This plastic re-operation cannot be satisfactorily performed if a vertical incision has been used. Furthermore, keloid formation is more common after a vertical incision.

In the beginning, only an incision was made in the trachea; but one patient with pulmonary complications after the closure of an atrial septal defect with the aid of a heart-lung machine was suffocated when the cannula slipped out and the inflated cuff compressed the trachea. Therefore, we always make a hole in trachea but, instead of excising a portion of the anterior wall of the trachea, this portion is left attached to the trachea inferiorly, constituting a flap of the tracheal wall fixed to the skin by a catgut suture (Fig. 1).

Advantages are (1) preventing the cannula from slipping down in front of the trachea below the tracheostomy; (2) the patient can breathe more easily through the opening in the trachea even in the first days if the cannula should become dislocated; and (3) if the flap is 4 to 5 mm. broad and has its base inferiorly, an open tracheostomy facilitates the exchange of the cannula. This flap has not caused excessive granulation tissue and stricture formation. On the contrary, if the cannula is not left in place too long the flap will fall back and prevent excessive formation of granulation tissue through the stoma.

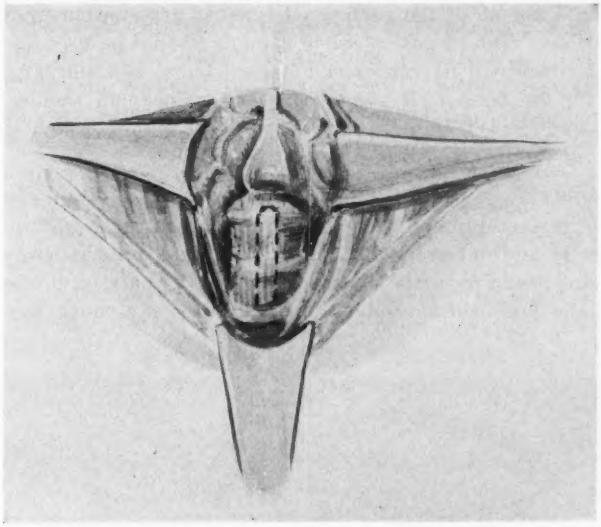
It is very important to make the hole in the trachea as high as possible, preferably through the second, third, and fourth cartilages (Fig. 1, *B*). In cases in which a low tracheostomy has been performed, it has been very difficult to keep the cannula in place and the connection airtight. The rubber cuff on the silver cannula may be partly outside the trachea if the tracheostomy is too low. When the cuff is inflated, the cannula may be pushed out of the trachea (Fig. 1, *C*). In one case in which the tracheostomy was too low, the cannula was repeatedly displaced when the cuff was inflated and the wound had to be packed with gauze around the cannula to make the connection somewhat airtight.

A firm fixation of the cannula by the band around the neck is especially important in children difficult to keep in the same position. This band can quickly be cut and the cannula removed if occluded.

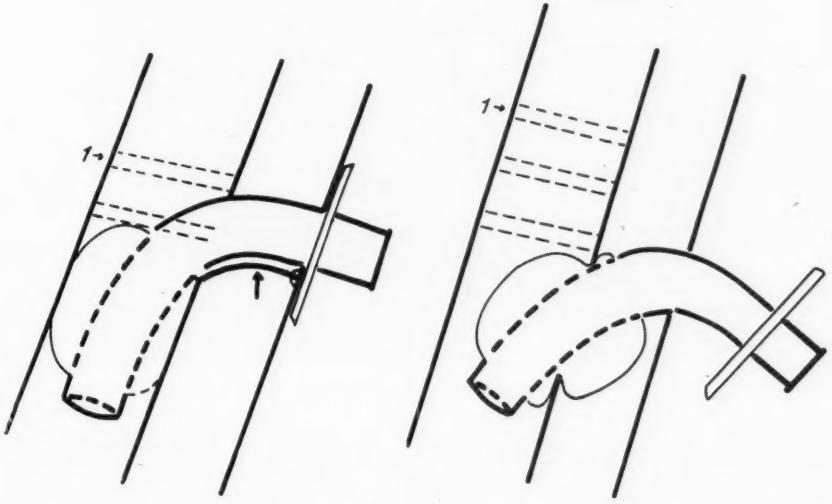
The Cannula.—

Rubber tubes should not be used due to the following disadvantages: (a) they are often too long, causing occlusion of one (the left) main bronchus; (b) they may not have the right curvature, causing pressure on the posterior membranous wall of the trachea with an esophago-tracheal fistula as a result; and (c) a rubber tube will soon be narrowed by dried up secretion.

A silver cannula, on the other hand, is easily kept open, as the inner cannula can be taken out daily for cleaning. We have never seen an esophago-tracheal fistula caused by a rubber-cuffed silver cannula. The silver



A.



B. *C.*

Fig. 1*A*.—Diagram of the incision in the trachea through the second and third cartilages with a flap of the anterior tracheal wall which may be sutured to the skin.

B, Diagram of the correct position of the cannula in the trachea. Observe the anterior tracheal flap (→) which will prevent the cannula being dislocated to the space in front of trachea; the flap will keep the stoma open and facilitate exchange of the cannula. With the high incision including the second down to the fourth tracheal rings, the rubber cuff will stay inside the trachea.

C, Diagram of the wrong position with a low tracheostomy and the rubber cuff partly outside the tracheal lumen, thus displacing the cannula outward when the cuff is inflated.

cannula should not be of the perforated type, as granulations may grow into the perforations. The largest possible cannula should be used for each case. In very small children, no rubber cuff is used but a cannula, large enough to practically fill the trachea, is employed. The rubber cuff should be as short as possible and placed close to the tip of the cannula, which then is kept more easily in the right position (Fig. 2).

The Respirator.—

A. The respirator must be volume cycled, that is, a certain amount of gas must go in and out from the lungs per minute quite independent of the resistance and pressure in the airway. If it works only on pressure and the patient coughs and works against the respirator not much gas will reach the alveoli.

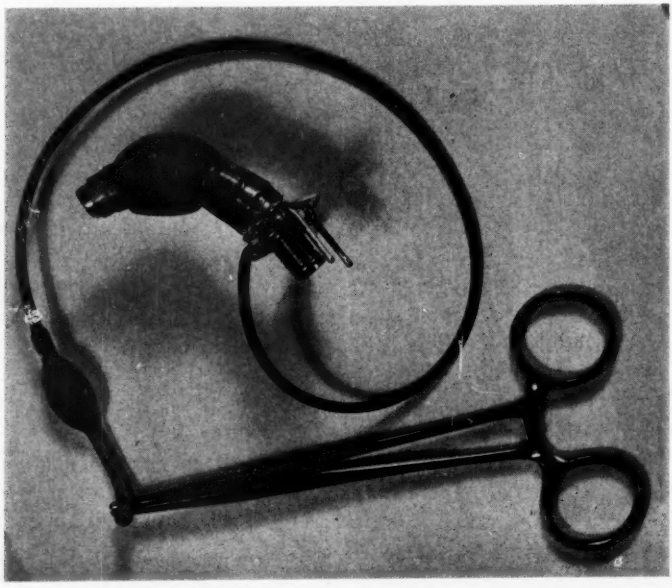


Fig. 2.—Photograph of the silver cannula with its rubber cuff.

B. Once a certain minute volume of ventilation is guaranteed, the respirator should be able to deliver this exact volume with any kind of pressure curve wanted for that special case in a semi-open system. When a patient with minimal functioning lung parenchyma is treated, the negative phase of the respirator is added, and its frequency increased. On the other hand, a ventilatory insufficiency due to pulmonary edema from mitral insufficiency is best handled when an expiratory resistance is added in the respirator. (Six centimeters of water pressure in expiratory resistance will immediately cause a pulmonary edema to disappear.)

C. What pressure curve is then ideal in the constant volume delivering respirator?

1. First, the inspiration must start from a pressure of zero and slowly build up the pressure. In other words, there must not be a sudden pressure explosion with danger of damage to the lung parenchyma. Then a terrific speed of the gas, 7.5 L. per second, is needed to move the normal tidal air in an elastic way without danger for the alveoli. The maximum flow is reached at the maximum pressure.

2. After the maximum pressure and flow comes a period of constant high pressure. "The plateau formation" equalizes the pressure in all areas of the lung and produces an even alveolar ventilation, even in the pressure of partial bronchial obstruction.

3. The expiration may be only passive, or a negative phase is most often added. At the end of expiration the air does not move and the pressure is zero. All credit goes to Engström for having constructed such a respirator, which is the only respirator so far I have seen or tested that fulfills all these criteria (Fig. 3).

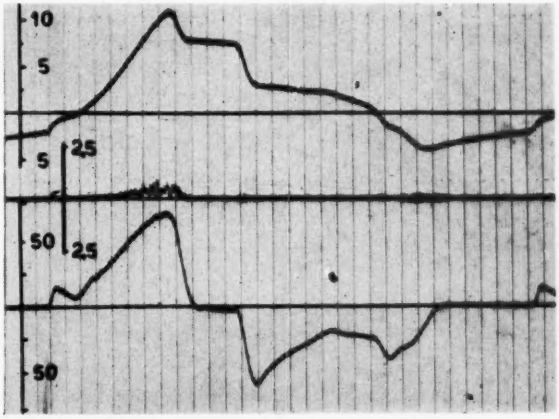


Fig. 3.—The *upper* curve shows the intratracheal pressure, the *lower* curve represents the flow utilizing an Engström respirator with negative phase during expiration.

METHOD

A. *Constant Ventilation.*—

If possible, the patient should be instructed about the details of the respirator treatment before it is instituted. The patient may then better follow the machine. There is usually no difficulty in continuing the respirator treatment directly after an operation. If, however, this treatment is instituted some days after the operation, it may sometimes be difficult to get the patient synchronized with the machine. When the patient works against the respirator either it should be disconnected or the valves should be closed, preventing the patient from inspiring air from outside the machine. Giving repeated doses of morphia every 4 hours relieves all pain as well as making it easy for the patient to synchronize with the machine which will take over

all the respiratory work. The frequency and minute volume must be adjusted to the size of the patient. An adult patient usually needs 10 L. ventilation per minute. A mixture of 50 per cent air and 50 per cent oxygen has proved to be satisfactory in most cases.

B. *Airtight Connection.*—

From time to time it is necessary to see that the ventilation is kept constant. The expired volume is measured, and if it is equal to the inspired amount, no leakage in the system is indicated. If the expired volume is less than the inspired volume, there is usually a leak around the rubber cuff on the silver cannula. At every inspiration some gurgling sound may be heard

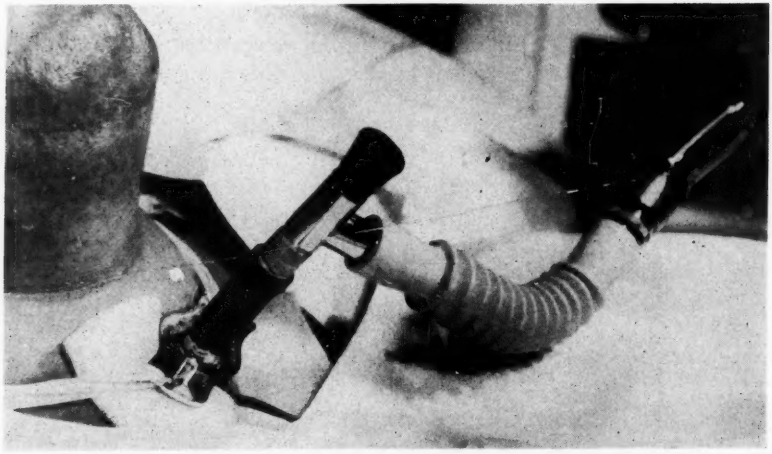


Fig. 4.—Photograph of the connection between the tracheal cannula and the respirator. The cork is removed for introduction of a catheter for aspiration.

as the gas passes the larynx and out through the mouth or nose instead of into the lung. Careful observation of and listening to the patient may disclose either that the rubber cuff is inflated too little or has ruptured and needs to be exchanged. Obviously the continous delivery of gas must be guaranteed.

C. *Continuous Gas Supply.*—

There have been two accidents, one with a lethal outcome, in which the oxygen supply was emptied while the patient was connected in an airtight manner to the respirator. Half the volume of air needed was not sufficient for a seriously ill patient during the time before detection of the error.

D. *Open Airway.*—

The trachea and the bronchi must be aspirated at intervals. The cannula is therefore connected to the respirator with a T-tube (Fig. 4). One need only remove a cork to introduce a catheter for aspiration. Secretions may

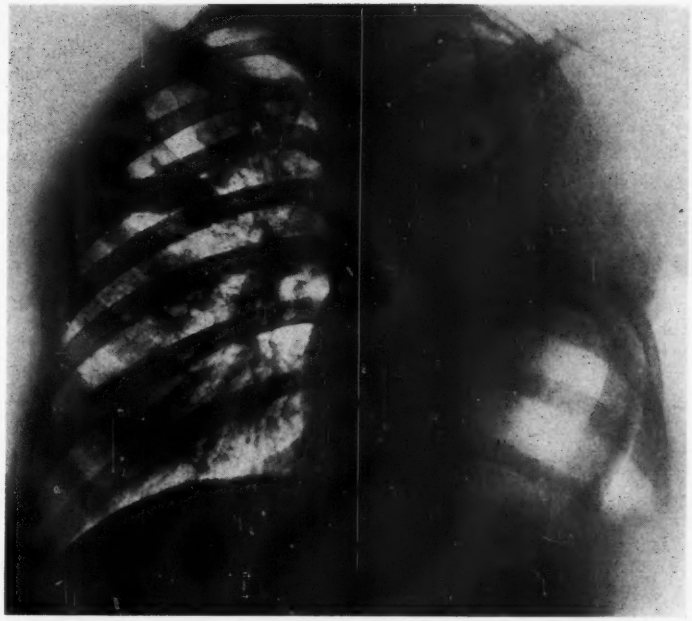


Fig. 5*A*.—Case 1. X-ray study of a 26-year-old woman having a giant cavity in the right upper lobe after a left pneumonectomy. Organisms were resistant to INH, PAS, and streptomycin. Cavity represents a completely destroyed right upper lobe.

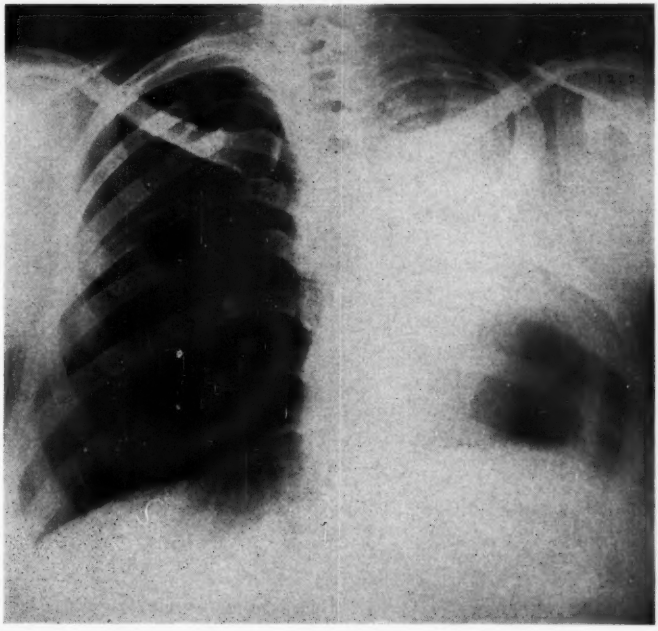


Fig. 5*B*.—Case 1. X-ray study made 1½ years after the operation.

dry, causing obstructions in the airway. The inner cannula should be removed for cleaning and boiling daily. The easy passage of the suction catheter shows that the cannula is in the right position and that it is not blocked. From time to time we have removed fibrin clots which filled the cannula. If the cannula is blocked by a clot that cannot be aspirated, the cannula must be removed quickly at which time the clot is usually dislodged with a cough of great force. If not, a bronchoscope is inserted for the removal of the obstruction. If dried up secretions are found, it is most important to inject 10 ml. of saline solution into the trachea. This should then be repeated at intervals.

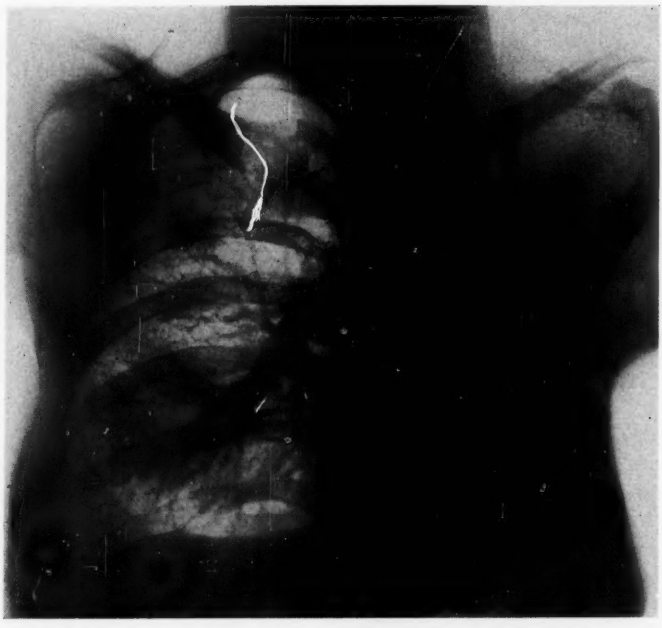


Fig. 6.—Case 2. X-ray study of a 29-year-old woman 3 years after left pneumonectomy and right upper lobectomy.

CASE REPORTS

The following cases with partial resection of the only remaining lung represent an indication for institution of respirator treatment immediately after surgery.

CASE 1.—A 26-year-old woman with bilateral pulmonary tuberculosis underwent left pneumonectomy in 1956. In 1957, a large cavity appeared in the right upper lobe (Fig. 5*A*). Organisms were resistant to isonicotinic acid hydrazide, para-aminosalicylic acid, and streptomycin (she had received more than 200 Gm. of streptomycin). A right upper lobectomy was performed in the face-down position, followed by 4 weeks of respirator treatment. The patient was then able to take over the ventilation herself and made

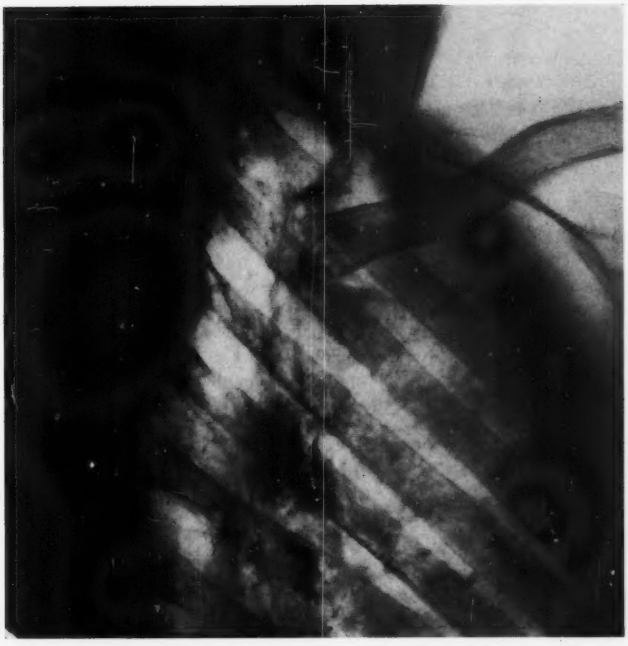


Fig. 7*A*.—Case 3. X-ray study demonstrating an apical cavity in the only remaining left lung.

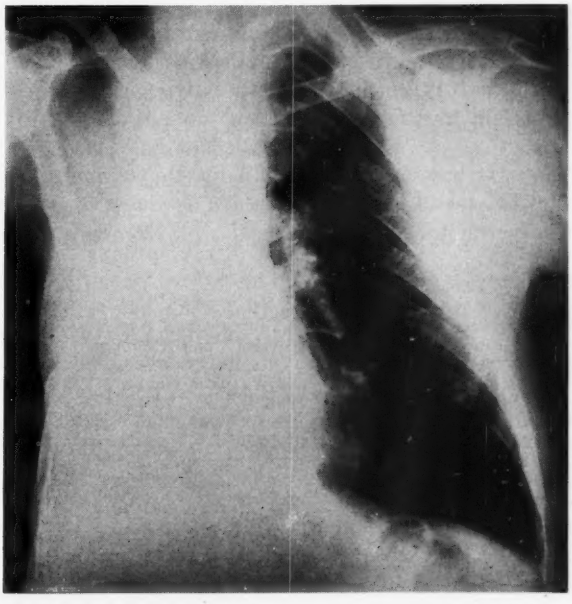


Fig. 7*B*.—Case 3. Roentgenogram made 4 years after resection of the apicoposterior and inferior lingular segment of the left upper lobe following a previous right pneumonectomy. Patient can walk three stairs without being short of breath.

an uneventful recovery. Two years after the operation (Fig. 5*B*) this patient's sputum was negative for tubercle bacilli. She had a normal sedimentation rate (7 mm.), and the vital capacity was 2,200 ml. She had no cough; she was no more dyspneic than before the operation but she felt very short of breath walking upstairs after having had a heavy meal. She worked all day with a 2-hour rest in the middle of the day. She cared for her home and two children, and did the cleaning and washing herself.

CASE 2.—A 29-year-old woman with bilateral pulmonary tuberculosis underwent left upper lobectomy in 1954 and right upper lobectomy in 1955. In 1956 she underwent a reresection of the left side resulting in a total pneumonectomy with an osteoplastic thoracoplasty, as the organisms were streptomycin resistant and she had a residual cavity. Postoperative respirator treatment was needed for 3 weeks. One year later she had a child by cesarean section and 3 years after the last operation she was doing housework, taking care of her child and husband. An x-ray study (Fig. 6) shows the well-functioning remaining two lobes of the right side. The maximal breathing capacity was 65 L. per minute and her sputum was negative. The sedimentation rate was 7 mm.

CASE 3.—A 29-year-old man underwent a righ pneumonectomy in 1954 for pulmonary tuberculosis. In 1955, a tuberculous cavity appeared in the apex of the left lung (Fig. 7*A*). He then underwent a resection of the apicoposterior segment and the inferior lingula segment of the left upper lobe. Postoperative respirator treatment was necessary for 12 days. The patient then made an uneventful recovery and 4 years after the operation he is working full time as a driver, and doing extra work in the evening, mounting television antennas on the roofs of houses. An x-ray study (Fig. 7*B*) shows no active tuberculosis. There is negative sputum, a normal sedimentation rate (2 mm.), and a maximal breathing capacity of 70 L. per minute with a vital capacity of 2,300.

SUMMARY

Partial resection of the only remaining lung for open tuberculous cavities can be performed with safety for the patient as long as tracheostomy with respirator treatment is given in the postoperative period. Patients lose some pulmonary function, but subjectively they do not notice this loss, as the sputum disappears at the same time. All 3 patients have gone back to an active life; one also having delivered a baby, the second able to look after her home and two children, and the third is doing full-time work. All sputa are negative for tubercle bacilli.

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STUDIES OF PULMONARY FUNCTION BEFORE AND AFTER PULMONARY RESECTION IN 450 TUBERCULOUS PATIENTS

II. Case Analysis of Patients With Large Loss of Vital Capacity and Maximum Breathing Capacity

John T. Mendenhall, M.D., Edna Cree, M.D., Howard K. Rasmussen, B.S., Helene Bauer, A.B., and John K. Curtis, M.D., Madison, Wis.

A previous communication[1] described the average loss of pulmonary function resulting from a variety of operations for removal of tuberculous lesions of the lungs. A close correlation was shown between the amount of functioning lung tissue removed and the loss of function, as measured 6 months postoperatively. There is generally little significant decrease of function in segmental and lobectomy operations. More extensive removal of lung tissue, such as bilateral resections and pneumonectomy, may cause rather large loss of function. In any series of resections, a number of complications may occur which are capable of profoundly altering pulmonary function. It is important for the physician referring patients for resection, as well as for the consulting surgeon, to know approximately how much function may be lost in the event of complications arising as a result of resection. It is the purpose of this paper to review the 450 tuberculous patients previously reported and to select for analysis those who had the greatest postoperative loss of vital capacity and maximum breathing capacity.

MATERIAL AND METHODS

The vital statistics and description of the methods used in testing the pulmonary function of the 450 tuberculous patients may be found in the first paper of this series. All patients received pulmonary function screening tests 1 to 2 weeks before resection and at an average time of 6 months after operation. It was shown that within this period most patients regain their maximum function. The patients who suffered the greatest loss of vital capacity and maximum breathing capacity were tabulated. The percentage loss of vital capacity and maximum breathing capacity were added together and will be designated as the combined loss. A cut off level for large combined losses was then selected which included a representative sample for patients receiving each type of operation, excluding pneumonectomy and decortication.

From the Surgical and Medical Departments, Veterans Administration Hospital, Madison, Wis., and the University of Wisconsin Medical School.

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RESULTS

One hundred and eighty-nine patients underwent unilateral segmental operations. Within this group were 9 patients who had an average loss of 18 per cent in vital capacity and 33 per cent in maximum breathing capacity, the maximum combined loss by a single patient in this group was 59 per cent. Lobectomy was performed in 123 patients, 7 had an average loss of 23 per cent in vital capacity and 36 per cent in maximum breathing capacity. The greatest combined loss in this group was 75 per cent. Fifty-three patients were subjected to bilateral segmental resections, 4 had an average loss of 35 per cent in vital capacity and 35 per cent in maximum breathing capacity. The greatest decrease in function in this group was found in 2 patients, each having 75 per cent combined loss. Review of patients undergoing bilateral lobectomy and segmental resection showed that there were 27 in this group, 7 had an average loss of 24 per cent in vital capacity and 33 per cent in maximum breathing capacity, the greatest combined loss was 62 per cent. Those having bilateral lobectomy only numbered 7 and there were 2 with average loss of 49 per cent in vital capacity and 49 per cent in maximum breathing capacity. In most of the patients there was a greater loss of maximum breathing capacity than of vital capacity.

TABLE I. COMPLICATIONS OF VARIOUS TYPES OF PULMONARY RESECTIONS RESULTING IN LARGE LOSS OF VITAL CAPACITY AND MAXIMUM BREATHING CAPACITY

	TOTAL NUMBER	NUMBER WITH LARGE LOSS	AVERAGE LOSS IN VC (%)	AVERAGE LOSS IN MBC (%)	SURGICAL COMPLICATIONS							
					BLEEDING	AIR SPACE	AIR SPACE THORACOPLASTY	ATELECTASIS	ATELECTASIS AND THORACOPLASTY	ADHESIONS OF DIAPHRAGM	DIAPHRAGM PARALYZED	NO COMPLICATION
Segments	189	9	18	33		2	3	1	1			2
Lobectomies	123	7	23	36	1*	1	1			1	1	2
Bilateral segments	53	4	35	35	1*		1			1	1	
Bilateral lobe + seg.	27	7	24	33			1*		1	1	2	2
Bilateral lobes	7†	2	49	49			1					1
Total	399	29			2	3	7	1	2	3	4	7

*Thorax reopened.
†43 pneumonectomies and 8 decortications not tabulated.

Table I shows the types of surgical complications which seemed primarily responsible for large loss of function in 29 of the 450 tuberculous patients. It is seen that there is a random scatter of complications among the various types of operations. No particular resectional operation can, therefore, be said to favor a specific complication. Intrathoracic bleeding occurred 13 times, requiring reopening of the chest to evacuate the clots and to ensure hemostasis. Extensive adhesions which resulted seem to be responsible for the loss of function in 3 of these patients.

Persistent air space may be defined as the roentgenologic appearance of loculated air usually at the apex, occasionally at the base, which persists

for more than a month postoperatively. It is postulated that a tiny air leak is responsible for such occurrences. If the air is replaced by fluid and pleural thickening or eventually absorbed without operative intervention it is usually not classified as a bronchopleural fistula. Three patients had this complication resulting in large loss of function, although at the time of testing the spaces had disappeared.

Preresectional and concomitant thoracoplasties are not performed at this hospital. In the patients having lobectomies in whom there are large spaces postoperatively, which seem unlikely to be filled by the remaining lung, it is customary to perform secondary thoracoplasty about 2 weeks following the resection. Eighteen lobectomies were followed by thoracoplasties in addition to the one tabulated in Table I. The average loss of vital capacity in the thoracoplasty group was 15 per cent compared with 10 per cent for the entire group. Maximum breathing capacity data showed identical results. Seven patients had persistent large air spaces following resection and required 3½ rib thoracoplasties for closure, resulting in large loss of function. There were 2 other patients in the segmental group who required secondary thoracoplasty and their change in function was only moderate.

Atelectasis occurring postoperatively is usually handled satisfactorily by bronchoscopy. Severe atelectasis which did not clear rapidly following bronchoscopy occurred in one case of segmental resection. Atelectasis requiring thoracoplasty also occurred in one segmental case and one bilateral operation on the side of lobectomy.

Fluoroscopy revealed extensive adhesions involving the diaphragm in 3 patients with large loss of function. Four patients were noted to have paralysis of the diaphragm on the operated side diagnosed by elevation of the diaphragm and a positive sniff test seen by fluoroscopy.

Seven patients in the series have no obvious complications to account for the large combined loss of function. All were in the older age group and 3 of these were noted to have diffuse emphysema. In the patients subjected to pneumonectomy, the loss of function is due primarily to resection of functioning lung tissue. This is especially apparent when the lung is removed for scattered disease rather than for destroyed lung, as was discussed in the previous communication. Space filling thoracoplasty also causes loss of function on the contralateral side.

DISCUSSION

A number of articles have appeared in the literature regarding changes in pulmonary function following pulmonary resection since the early paper of Cournand and Berry.[2] Preoperative and postoperative values are now available for wedge resections, segmental resection, lobectomies, bilateral resections, and pneumonectomies. Only a few authors mention the importance of complications and apparently no papers are devoted primarily to complications leading to maximum loss of function.

Kraan,[3] in discussing resection in tuberculosis, draws attention to the importance of the intact diaphragm in preserving function. Phrenic avulsion or damage to the nerve during operation causing paralysis of the diaphragm may lead to marked loss of function. Van der Drift[4, 5] came to similar conclusions. The complication of activation of tuberculosis in portions of the lung not removed is cited by Kraan,[3] but in our experience this is a rare occurrence and has not greatly affected pulmonary function within 6 months after surgery. Such complications as bronchopleural fistula and empyema are frequently mentioned as causes for loss of function but these are not documented. A recent article by Björk[6] lists the following complications of segmental operations without correlating loss of function: (1) air leakage, (2) accidental opening of a tuberculous cavity, (3) wound infection, (4) ventilatory insufficiency, (5) trauma to big vessels, (6) postoperative pain, and (7) spillover with infiltration in the contralateral lung. Complications such as persistent air space, air leakage, bronchopleural fistula, atelectasis of the lung, and empyema are frequently corrected by thoracoplasty. Gaensler,[7] Lindahl,[8] and Little[9] have shown that this procedure is apt to cause a further loss of function. On the other hand, plombage for filling uninfected spaces causes a lesser diminution of function.[7, 9]

It should be emphasized that Table I does not include all the surgical complications that occurred in these 450 tuberculous patients, but only those which appeared to be associated with large loss of function. Furthermore, there were occasional deaths. Some patients severely ill from empyema or bronchopleural fistula were not available for retesting at the 6-month period. Statistics on a comparable series, including many of these patients, will be found in the Transactions of the 18th Conference of the Chemotherapy of Tuberculosis, Veterans Administration Armed Forces, 1959.

The development of postoperative paralysis of the diaphragm is worthy of special mention. In none of our 4 patients with this complication was the phrenic nerve cut or stretched at the time of operation. Great care was taken not to use the cautery to cut adhesions or control bleeding in the area traversed by the phrenic nerve. The cause for the development of paralysis remains unexplained. We have observed 1 patient, not in this series, who postoperatively had a paralysis of the diaphragm on the side opposite the operation which would seem to preclude direct injury of the phrenic nerve. That the loss of function is large, following paralysis of the diaphragm, is shown by the 4 patients in Table I; the combined loss for each was 61, 75, 57, and 56 per cent.

SUMMARY

Four hundred and fifty tuberculous patients received preoperative and postoperative pulmonary function tests. Twenty-nine patients had large losses in the vital capacity and maximum breathing capacity following operation. These cases were analyzed to determine the types of surgical complications that occurred. Intrathoracic bleeding or persistent air spaces leading to

pleural thickening and adhesions, protracted atelectasis, and impaired diaphragmatic motion were found to be often associated with a significant reduction of function. Complications requiring secondary thoracoplasty also resulted in large loss of vital capacity and maximum breathing capacity. In 4 patients, idiopathic paralysis of the diaphragm seemed responsible for impairment of ventilatory function.

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RESULTS AND COMPLICATIONS OF PLEURECTOMY FOR BULLOUS EMPHYSEMA AND RECURRENT PNEUMOTHORAX

Paul A. Thomas, Captain, MC, USA, and Paul W. Gebauer, M.D.,***
Honolulu, Hawaii

SUBTOTAL excision of the parietal pleura for the treatment of recurrent pneumothorax and giant bullous emphysema has been reported by other surgeons.[1-4] We have reported our experiences in a series of patients with pneumothorax.[5] In this paper, the results and complications of pleurectomy for pneumothorax are augmented by additional patients and the results with patients treated for bullous emphysema are recorded. Between August, 1951, and June, 1958, 41 patients had 45 (4 bilateral) operations.

Of these patients, 28 were either in the military service, dependents of active duty service personnel, or were veterans who qualified for admission and treatment on the Surgical Service at Tripler U.S. Army Hospital. An additional 13 patients were seen in the thoracic surgical practice of one of us (P. W. G.). The indication for surgery was recurrent spontaneous pneumothorax in 27 instances and in the remaining 18 was bullous emphysema. The average age at the time of surgery for the patients with pneumothorax was 31 years, with a range of 19 to 71 years. For those patients with bullous emphysema the average age was 39 years at the time of surgery with a range of 28 to 70 years.

We consider subtotal parietal pleura excision the most certain method of pleural space obliteration and superior to the instillation of irritants or other open methods.

The first patient in this series was operated upon in 1951 for recurrent spontaneous pneumothorax. The second patient, for whom parietal pleurectomy was employed, submitted to staged, bilateral operations for giant bullous emphysema. The excellent result obtained in these 2 patients was encouraging and pleurectomy has since been incorporated as an important component of our surgery for these two conditions.

In addition to the obvious indication for pleurectomy, obliteration of the pleural space, a secondary gain may be realized by revascularization of the lung. Barrett and Daley[6] reported the feasibility of providing increased blood

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*Present address: US Army Surgical Research Unit, Brooke Army Medical Center, Fort Sam Houston, Texas.
**Thoracic Surgical Consultant, Tripler US Army Hospital.

supply to the lung from the depleuralized chest wall for the patients with tetralogy of Fallot technically unsuitable for the Blalock or Potts operations. Crenshaw and Rowles[4] attempted surgical revascularization of the atrophic degenerated lungs of emphysematous patients by this method. The gross pathologic changes in the lungs of patients with spontaneous pneumothorax as seen at the operating table are remarkably similar to those observed in patients with bullous emphysema in many instances. The single remarkable difference is the degree of involvement which is quite variable in any given patient. We believe that spontaneous pneumothorax may be the earliest clinical expression of an atrophic, degenerative alteration of the lung parenchyma which will be recognized in later life as emphysema. Therefore, if the fundamental pathophysiology of emphysema is a diminished peripheral pulmonary vascularity, progression might be prevented by early pleurectomy.

The patients reported in this experience were accepted as candidates for surgery with the following indications:

1. Spontaneous pneumothorax with persistent air leak (one patient).
2. Spontaneous pneumothorax with bullous lesion demonstrable by chest roentgenogram (7 patients).
3. Spontaneous pneumothorax, recurrent, unilateral, or alternating bilateral (17 patients).
4. Spontaneous pneumothorax, simultaneous bilateral (one patient).
5. Giant emphysematous bullae with progressive compression of functioning lung tissue (7 patients).
6. Giant emphysematous bullae symptomatic (8 patients).

In addition to excision of the pleura, surgery included lung repair by unroofing and oversewing surface blebs and bullae with minimal sacrifice of functioning lung tissue. Resection of destroyed bronchopulmonary segments or an entire lobe was necessary in only 4 of 45 operative procedures and is to be avoided as much as possible to preserve maximum function. Also, resection of the pulmonary plexus of the vagus nerve was recently employed in 4 patients who had symptoms of bronchospasm in addition to pulmonary emphysema.

Evaluation of postoperative results is based on observations which vary from 6 months to 6 years. Three patients died either directly or indirectly as a consequence of surgery. The 38 surviving patients were doing well when last seen. Of the 25 patients followed after surgery for spontaneous pneumothorax, 5 have been observed for 2 and 3 years. There have been no known recurrences of pneumothorax on the side of surgery. Spontaneous pneumothorax of the contralateral lung has occurred in 7 patients since surgery. Three of these patients had episodes of pneumothorax bilaterally, either alternating (2 patients) or simultaneous (1 patient) prior to surgery. One of the patients subsequently submitted to a second operation to stabilize the second lung; others may be candidates for further surgery. Of the group of patients treated for bullous emphysema, 5 have been followed 2 years or longer. The postoperative observation extends 5, 5½, and 6 years, respectively, for 3 of these patients. They are all symptomatically and subjectively improved. In two instances, pain was a primary symptom and this has been relieved.

A truly objective measure of the status of these patients has not been possible because complete bronchospirometric studies were not done. Postoperative roentgenograms of the chest appear essentially normal in the majority of patients with minimal residual scarring in a few. Evaluation of preoperative and postoperative pulmonary function has been extensively investigated following surgical obliteration of the pleural space by other investigators.[1, 7] Selected patients were evaluated either because of some interesting aspect of the postoperative course, or as a long-term follow-up examination. The results of vital capacity and maximum breathing capacity measurements are presented

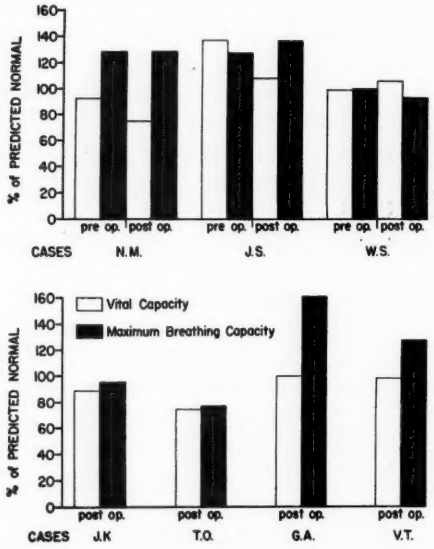

Fig. 1.—Pulmonary ventilatory function studies.

in Fig. 1. Patient N. M. underwent right thoracotomy for giant bullous emphysema. There was little change in ventilatory function observed 2 years after surgery as compared with studies done prior to operation. The degree of intrapulmonary mixing was normal, as well as measurements of residual volume. Patient J. S. also revealed little change in ventilatory function after surgery. These studies were of particular interest because the patient had a complicated postoperative course with hemothorax, empyema, and required a delayed second thoracotomy for decortication some months after the pleurectomy which was done for bullous emphysema. Here again intrapulmonary mixing and residual volume determinations were normal and unchanged by surgery. The next patient, W. S., was operated upon for recurrent spontaneous pneumothorax with associated bullae demonstrable by chest roentgenogram. Once again there is amazingly little change noted in the postoperative measurements as compared with preoperative values. Patient J. K. submitted to pulmonary function

studies 6 years after bilateral surgery for giant bullous emphysema. Preoperative studies are not available for comparison. Vital capacity and maximum breathing capacity are slightly less than predicted normal values. However, this patient required resection of more lung tissue than any other in the series; the apical and posterior segments of the right upper lobe and the entire left upper lobe were removed. The next patient, T. O., was studied 5 years after bilateral surgery for giant bullous emphysema. The second operation was complicated by the development of empyema. Pulmonary function was significantly impaired in this individual. In addition to decreased vital capacity and maximum breathing capacity, the patient had paradoxical motion of the right diaphragm and increased residual air volume. The final 2 patients, G. A. and V. T., were studied approximately one year after surgery had been performed for recurrent spontaneous pneumothorax and giant bullous emphysema, respectively. The measurements are significant only in that no grossly demonstrable residual impairment is evident. Our observations of pulmonary function indicate that parietal pleurectomy, in the absence of complications, does not appear to impair the ventilatory capacity of these patients.

Some of the most important observations to be made from this series of cases concerns the complications of surgery. The operations at Tripler U.S. Army Hospital were performed for the most part by the resident staff of the surgical service. In 24 instances the operative procedures were done by one or the other of the authors; the remainder were performed under the supervision of the thoracic surgical consultant (P. W. G.). There have been 3 deaths in this group of patients since surgery, a gross mortality of 7 per cent. Two of these deaths occurred in the immediate postoperative period and one 10 weeks following surgery.

In the first case that ended fatally, surgery was undertaken in an effort to salvage a moribund patient. This individual was 71 years of age and had marked, crippling pulmonary emphysema. In addition, he had suffered repeated episodes of life-endangering pneumothoraces over the last 2½ months of life, which required intercostal catheter decompression of the pleural space and tracheostomy to prevent total respiratory collapse. He was also on digitalis for cardiac decompensation. Surgery was undertaken, with reservation, because of increasing dyspnea in spite of therapy. The patient rapidly failed after surgery in spite of mechanically assisted respiration.

The next patient was 60 years of age with marked bullous emphysema and pulmonary insufficiency. Following operation, the immediate postoperative course was uneventful and the patient was discharged from the hospital on the twelfth day after surgery. He returned to be readmitted to the hospital on the eighteenth postoperative day with signs and symptoms of acute, fulminating renal failure. This patient died within 2 days in spite of therapy and at postmortem examination a lower nephron nephrosis was observed.

The third fatal case was remarkable in that the postoperative course was complicated by many factors and extended over a 2½ month period. This patient was 37 years of age and was apparently in good health at the time

of the operation. The immediate postoperative course was complicated by hemorrhage of moderate severity over a period of 5 days. Blood loss was not massive during this time but the patient required transfusions daily. Subsequently, the patient recovered slowly to become asymptomatic with the exception of a slight exertional dyspnea. Repeated thoracenteses were done but all residual air pockets and blood could not be removed from the pleural cavity. The patient was allowed to return home to be followed at frequent intervals as an outpatient. Successive chest roentgenograms showed progression of the pneumothorax space and increased pleural reaction. The patient was readmitted 2 months following surgery for elective decortication of the lung. However, because of increasing dyspnea, anemia, and subsequent fever while in the hospital, surgery was withheld. Thoracentesis yielded purulent material. Within several days, fresh bleeding was detected within the pleural space, coincident with the development of a shower of cutaneous petechiae. Within a very short time, ecchymoses, and gastrointestinal hemorrhage developed and the patient became comatose and died. Postmortem examination confirmed the terminal clinical picture of thrombocytopenic purpura of undetermined origin. In retrospect, the initial postoperative hemorrhage was probably an unrecognized manifestation of the abnormality although the patient had a normal bleeding and clotting time prior to operation.

Any discussion of parietal pleurectomy always stimulates a question as to the amount of blood lost from the large denuded area of the chest wall. With the exception of the case just cited this has been a contributing factor in the increased morbidity of one additional patient only. This individual required 3,500 c.c. of whole blood transfusion during and after surgery. He developed a hemothorax which became secondarily infected. The patient subsequently submitted to a secondary thoracotomy and decortication. Pulmonary function studies one year later revealed little change (Fig. 1, Patient J. S.) as compared with those done prior to surgery. The blood replacement was tabulated and found to vary between 500 to 3,500 c.c. per patient, excluding the patient with thrombocytopenia who required well over 10,000 c.c. during the postoperative period up to his death. The average blood replacement during and following surgery, therefore, was 1,300 c.c. In only 5 instances was more than 2,000 c.c. of blood replacement required. The amount of blood replacement following complete parietal pleurectomy is comparable to that amount of blood necessary following most intrathoracic surgical procedures, and is not considered excessive. From this experience, it would be wise to consider all possible conditions which might contribute to hemorrhage in the postoperative period before accepting pleurectomy as the responsible factor in any given patient with excessive bleeding.

The complications encountered in this series of patients have been tabulated (Table I). It is interesting to note that the minor complications occurred primarily in the patients treated for pneumothorax as opposed to the group of patients operated upon for bullous emphysema in whom the major complications predominated. Any postoperative air leak beyond 5 days was

considered persistent. The longest period of postoperative air leak was 10 days; all subsided with continued intercostal catheter drainage. Similarly, any residual air space discernible by chest roentgenogram after removal of the thoracotomy drainage tubes was recorded as a residual pneumothorax.

TABLE I. COMPLICATIONS OF SURGERY

COMPLICATIONS	NO. CASES
Minor	
1. Persistent air leak	3
2. Residual pneumothorax	3
3. Contralateral pneumothorax	3
Major	
1. Hemorrhage	2
2. Empyema	2
3. Atelectasis	1
4. Lung abscess	1
5. Lower nephron nephrosis	1
6. Thrombocytopenia, idiopathic	1

All residual air spaces resolved spontaneously, usually within a short period of time. The longest time required for resolution was 4 months in one patient. Contralateral spontaneous pneumothorax was recorded as a complication only if it occurred during the postoperative period of hospitalization. In only one instance was this sufficiently severe to require treatment. The major complications, with the exception of the single episode of atelectasis, occurred in the group of patients treated for bullous emphysema. This is, in part, a reflection of the older age of these patients as well as the more extensive pulmonary disease present at the time of surgery. The eight major complications recorded were encountered in 5 patients following 5 operative procedures. Several of these have been discussed at some length. In addition to the patient with hemothorax who developed empyema, infection occurred in one additional patient following the second of staged bilateral operations. This was treated successfully by repeated aspiration and chemotherapy. Pulmonary function studies 6 years later in this patient (T. O.) revealed some impairment of ventilatory capacity as previously noted (Fig. 1). One patient returned to the hospital on the thirteenth postoperative day, after being discharged, with a small, aspiration-type lung abscess in the left lower lobe. A good therapeutic response to chemotherapy was obtained, with complete resolution in 7 days.

DISCUSSION

Partial parietal pleurectomy has a definite place as a method of obliterating the pleural space in patients requiring surgery for spontaneous pneumothorax and giant bullous emphysema. The immediate results following surgery have been gratifying in this series of patients. Several patients have now been followed 5 to 6 years without evidence of progression of lung bullae of a size sufficient to be discernible by chest roentgenogram. It is possible that

the additional increase in blood supply provided to the lung surface by this procedure will be of lasting benefit. In the absence of severe complications there is no indication that pulmonary function is jeopardized by this operation. The mortality following operation was not directly the result of pleurectomy although surgery was a contributing factor in each instance. If one continues to accept elderly debilitated patients with pulmonary insufficiency as candidates for surgery, this will be a deterring feature. Postoperative bleeding is not usually a major feature; however, when it does occur, it should not be too hastily attributed to pleurectomy lest one overlook other possible causes of hemorrhage. The usual blood loss associated with pleurectomy is comparable to that of other intrathoracic operations and has not been excessive.

The total incidence of postoperative complication was 38 per cent of 45 operations including 3 fatalities: Minor complications which did not influence or prolong morbidity constituted over one half of the total or an incidence of 20 per cent. In only one instance was a complication of surgery responsible for residual impairment of pulmonary function. Major complications, which included hemorrhage, empyema, atelectasis, and lung abscess, occurred in 5 of 38 surviving patients who submitted to 42 operative procedures, an incidence of 12 per cent. The high rate of surgical complications in this series should serve to caution those individuals who would recommend simultaneous bilateral surgery for patients with recurrent spontaneous pneumothorax as has been advocated.[2]

Although the pathologic changes may be bilateral in a significant number of patients with spontaneous pneumothorax, only 7 of 26 patients have had clinically detectable pneumothorax of the contralateral lung since operation. This might have been anticipated in 3 patients who had episodes of contralateral pneumothorax prior to surgery. On the other hand, 2 patients who had contralateral pneumothorax at some time prior to surgery have not had recurrence since surgery. Therefore, only 25 to 35 per cent of patients requiring thoracotomy for recurrent pneumothorax will have bilateral difficulties. Of these, not all will subsequently require a second operation. If all patients are subjected to simultaneous bilateral surgery, the larger percentage will have a needless or meddlesome operative procedure. The increased risk associated with such an undertaking does not seem to be justifiable at the present time.

SUMMARY

The following observations have been made from this series of 41 patients who submitted to 45 thoracic surgical procedures which included partial parietal pleurectomy as an important component of the operation. Pleurectomy will obliterate the pleural space and in addition may provide a desirable increase in blood supply to the peripheral lung tissue. Postoperative results are excellent in patients now observed up to 6 years following surgery. A significant mortality may be anticipated following surgery of this nature performed on individuals with marked pulmonary insufficiency or among those

in older age groups. Postoperative bleeding of alarming magnitude should not be hastily attributed to the raw pleural surface without first considering the possibility of rupture of a major blood vessel or some defect in the blood clotting mechanism as a causative factor. The incidence of surgical complications is of such magnitude and seriousness as to discourage any ill-conceived idea that simultaneous bilateral surgery of this nature is not fraught with great risk to the patient.

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SUPERIOR VENA CAVAL REPLACEMENT WITH SPONGE TEFLON PROSTHESIS

A Preliminary Report

Richard J. Botham, M.D., Theodore T. Dracopoulos, M.D., and Joseph W. Gale, M.D., Madison, Wis.

THE MANAGEMENT of superior vena caval obstruction has remained a singularly perplexing problem to the internist and vascular surgeon alike. The literature review of McIntire and Sykes,[1] published in 1949, contained 524 cases of superior vena caval obstruction. In 1952, Veal and Cotsonas[2] reported their experience with sixty-one obstructions which involved either the superior vena cava or the innominate veins. The 61 cases in their series had been seen over a 15-year period at one institution. Failor, Edwards, and Hodgson[3] recently reviewed 33 necropsy cases of superior vena caval obstruction, all of which had been seen at the Mayo Clinic. Allansmith and Richards[4] reported, from a single institution, a series of 21 cases of superior vena caval obstruction in which attempts at surgical alleviation had been undertaken. The above-mentioned reviews indicate that superior vena caval obstruction is not an infrequently occurring entity.

Primary malignant thoracic tumors are unquestionably the most common etiologic factor underlying superior vena caval obstruction.[1, 3] However, in 17 of the 21 surgically treated cases reported by Allansmith and Richards,[4] the underlying etiologic lesion was noted to be of a benign nature. The incidence of chronic fibrous mediastinitis as a primary etiologic factor in superior caval obstruction has been variously reported as 12 to 15 per cent.[1, 3] The latter lesion has an exceedingly poor ultimate prognosis if left untreated. It, therefore, behooves us to seek and develop a satisfactory means of surgical management for superior vena caval obstruction secondary to a benign causative factor. A consideration of the underlying disease process seemingly makes bypass or actual resection and replacement of the involved caval segment, perhaps, the most logical, as well as feasible, surgical procedure.

Numerous procedures have been devised and applied experimentally and, on occasion, clinically. Atriocaval anastomosis, described by Gerbode and associates[5] in 1949, and the use of a pedicle tube created from the right atrium and atrial appendage, described by Riberi and Moore,[6] have both been reported

From the Cardiovascular Research Laboratories and the Department of Surgery of the University of Wisconsin Medical School, Madison, Wis.

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as successful procedures when applied experimentally. Neither of these procedures appealed to us when considering the technical problems of their clinical application since they are both limited with respect to the length of superior vena cava which can be replaced. Autogenous aorta has also been used experimentally for superior vena caval replacement with a high incidence of success by Moore and Riberi.[7] The magnitude of such a procedure, and its probably significant associated morbidity and mortality, has discouraged us from consideration of this procedure as clinically applicable.

Deterling and Bhonslay[8] utilized autogenous venous tissue, enlarged through the panel technique, as a means of experimental superior vena caval replacement but found its use attended with a high incidence of failure. Aorta homografts and various synthetic prostheses have been applied experimentally[8, 9] and have also been found to be unsuitable as venous substitutes. Allansmith and Richards[4] reported various clinical successes with the use of arterial homografts and heterografts, venous autografts, and a crimped nylon prosthesis. They neglected to report the duration of follow-up in their cases and stated that some of the grafts were thought to have become obstructed after several months.

In spite of the above-mentioned reports of failure attending the use of synthetic vascular prostheses in superior vena caval replacement, they seemed to us to be the most appealing, logical, feasible, and applicable surgical procedure. Such a procedure is obviously dependent upon the development and utilization of a synthetic material whose physical and chemical properties have minimal associated host reaction and a high incidence of success as a vascular substitute.

We feel that these characteristics are inherently embodied in Teflon which, to date, has enjoyed considerable success as a vascular prosthesis in aortic and arterial replacement and bypass procedures. The purpose of this report is to present our experience with the use of porous sponge Teflon, constructed in the form of a vascular prosthesis, and its evaluation in experimental superior vena caval replacement.

MATERIALS AND METHODS

Healthy mongrel dogs, weighing 17 to 30 kilograms, were used in this study. Anesthesia was induced with intravenous Nembutal in a dose of 30 mg. per kilogram of body weight. Respiration was maintained with the aid of a cuffed endotracheal tube, and a mechanical respirator utilizing alternately positive and negative pressure.

In all animals, a right thoracotomy was performed through the right third intercostal space. The vena cava was dissected free from its attachment to the right atrium to include the right and left innominate veins. The right innominate vein was ligated and divided to allow excision of as long a segment of vena cava as possible. The length and circumference of the segment of vena cava to be excised were measured. A graft of slightly larger dimensions

was constructed from a ⅛ inch thick sheet of porous sponge Teflon. The longitudinal seam of the graft was doubly sewn with an over-and-over suture of 3-0 vascular silk in such a manner that the sutures were not visible within the lumen of the graft. That segment of the vena cava extending from just distal to the left innominate vein to just proximal to the azygos vein was excised and replaced with the previously constructed prosthesis of porous sponge Teflon. A single row of over-and-over 5-0 vascular silk sutures was used for both anastomoses. Vena caval occlusion during the period of resection and anastomosis was maintained by the use of atraumatic curved clamps. The period of vena caval occlusion varied from 28 to 33 minutes. At the completion of the procedure, the graft was not covered with pleura (Fig. 1). The

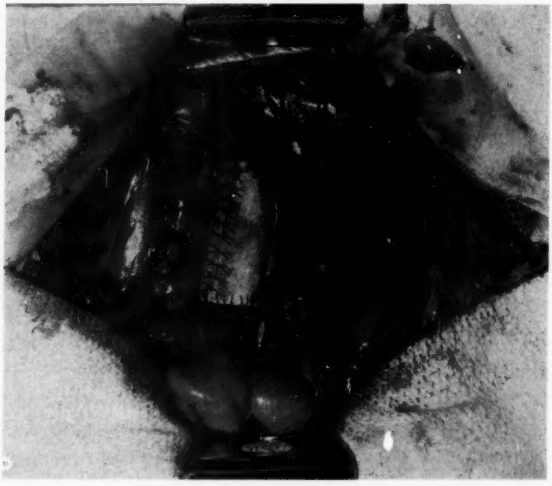


Fig. 1.—Porous sponge Teflon prosthesis replacing the superior vena cava. Longitudinal seam of constructed graft readily apparent. Phrenic nerve along left side of graft.

chest wall was closed in layers and a catheter was left in the right pleural space for 24 hours for aspiration of air and drainage material. Penicillin was given the day of and the day subsequent to operation.

RESULTS

The operation of superior vena caval replacement with a prosthesis constructed from a sheet of porous sponge Teflon was carried out as described in 20 dogs. Maintained patency of the prostheses was determined in all animals by superior vena cava angiogram, autopsy, or both, from 1 week to 6 months following operation.

Four animals died at periods of 1 week to 2 months following the operative procedure. Atelectasis was the apparent cause of death in a single animal which succumbed 1 week following operation. Autopsy in this case revealed a patent graft. A second animal died apparently from affects secondary to

occlusion of the graft as determined at the time of autopsy. A third animal succumbed to a respiratory infection and autopsy in this case revealed a patent graft. The cause of death in the last animal was not apparent at the time of postmortem examination and the graft in this case was also found to be patent.

Superior vena cava angiograms were performed on 16 animals. Twelve of the grafts were found to be patent for varying periods of 1 to 6 months (Fig. 2). Four of the prostheses were noted to be occluded and this finding was substantiated by subsequent animal sacrifice and autopsy. In 2 animals, although definite patency of the grafts was established, there was noted to be a narrowed segment within the graft. The latter animals have remained healthy and will

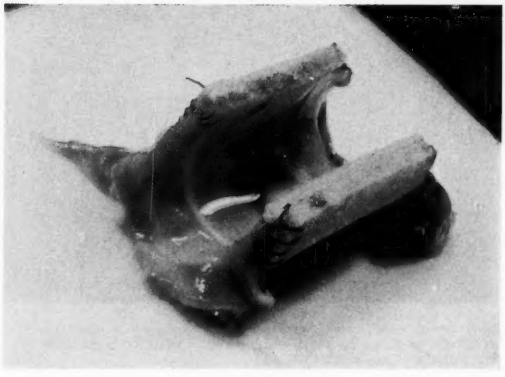
Fig. 2.—Superior vena cava angiogram of an animal 4 months following insertion of porous sponge Teflon graft in the superior vena cava. Widely patent graft apparent with good visualization of right auricle and ventricle.

ultimately undergo further evaluation to determine duration of graft patency and host reaction to the porous sponge Teflon prostheses. A single animal was sacrificed 2½ months following operation to corroborate findings of patency, as previously determined by superior vena cava angiogram, and to allow gross and histologic evaluation of the fate of the prosthesis and the associated host tissue reaction. In this latter case, a widely patent functioning graft was found (Fig. 3). Eleven animals with patent grafts remain well and will be maintained to allow ultimate long-term studies of duration of graft patency and gross and histologic host tissue reaction.

Graft patency, then, was maintained, as determined by superior vena cava angiogram and/or postmortem examination, in 15 of the 20 animals in the study for a maintained patency percentage of 75 per cent.

The appearance of the graft lumen in the 4 animals who died or were sacrificed and had patent grafts varied with the duration of time subsequent to the operative procedure. The lumen of the graft obtained 1 week following

A.



B.

Fig. 3.—Sponge Teflon superior vena caval prosthesis removed from animal 2½ months following surgery. *A* shows graft on end with everted segment of vena cava and smooth lining of graft. *B* shows same graft opened with smooth lining at sites of anastomosis and within the graft lumen.

surgery was noted to be lined with an adherent, fibrinous coagulum approximately 2 mm. in thickness (Fig. 4). The lumens of the grafts examined 2 weeks following surgery had a similar adherent, fibrinous lining, which in areas appeared to be less than 2 mm. in thickness as though the lining was undergoing a process of resorption in those scattered areas. The lumen of the graft examined 2½ months subsequent to surgery was noted to have a

smooth, shiny, thin lining as though completely endothelized (Fig. 3). The suture lines in all four patent grafts examined were well healed as early as 1 week following surgery (Fig. 4).

Postmortem examinations performed on animals, after a period of a month or greater had transpired since surgery, revealed densely adherent fibrous tissue reactions about the grafts. Although the fibrous tissue about the graft could be stripped from the external surface, it appeared to be intimately adherent as though invading the graft proper. Histologic studies of canine host reaction to implanted porous sponge Teflon, carried out prior to and in conjunction with this study, revealed to us that the sponge Teflon is actually invaded by host fibroblastic reaction and is well incorporated into the body by the host fibrous tissue reaction. A more detailed description of canine host tissue response to implanted porous sponge Teflon will appear in a subsequent report.[10]

Fig. 4.—Porous sponge Teflon prosthesis removed from animal one week following surgery. The graft was patent and functioning. Thin adherent fibrinous coagulum is seen lining the graft lumen. Good healing seen at sites of anastomosis.

DISCUSSION

Certain features inherent in synthetic vascular prosthesis which make them highly desirable are worthy of review. Among these are their ready availability in varying sizes. The sponge Teflon used in this study was obtained in flat sheets, ⅛ inch thick, which were malleable enough to allow construction of varying sized tubes. The grafts, as we constructed them, were admittedly somewhat crude and of necessity contained a longitudinal seam. Due to the fact that the material was ⅛ inch thick, we were, with some practice, ultimately able to construct a prosthesis without the suture material presenting within the lumen of the graft. We hoped that this would lessen the chance of initiating clot formation at the site of the longitudinal seam. Seamless tubes of the same material in varying sizes would obviously be more desirable than our somewhat crudely constructed grafts.

A highly desirable characteristic of Teflon is the fact that it may be autoclaved without significant alteration of its physical properties. To

ensure asepsis, all of the Teflon used in these experiments was autoclaved prior to construction and implantation of the prostheses. The physical characteristics of the sponge Teflon, in the thickness used, allowed a significant degree of firmness of the grafts and prevented collapse of the implanted prostheses, an obviously desirable feature when considering the problems of a vascular substitute for replacement in such a low pressure system as the vena cava. A graft which is easily collapsible in a low pressure system, or readily compressible by a normal fibrotic tissue response, has obvious liabilities as a venous substitute and was therefore avoided.

The excellent experimental and clinical results of others with the use of Teflon prostheses as arterial substitutes appeared to us an additional reason for experimenting with its use in the venous system. Although we were initially concerned with the host reaction to Teflon in the sponge form, implantation studies, previously alluded to, revealed to us that the material would, in fact, ultimately be invaded by host fibroblasts and be incorporated within the host fibrous tissue reaction. The porous sponge Teflon contains interconnected spaces which allow growth of fibroblastic tissue into and ultimately completely through the prosthesis, a factor apparently necessary in any synthetic material if it is to be accepted by the host as a vascular substitute. The incidence of successful vena caval replacement with a synthetic substitute, which was achieved in these studies, stands in sharp contrast to the rather high incidence of failure with the use of other synthetic prostheses similarly employed and previously reported by others.[8] Although excellent results have reportedly accompanied the experimental use of fresh autogenous aorta as a vena caval replacement, its use entails a far more formidable procedure than the use of a readily available synthetic prosthesis. If, then, similarly excellent results can be obtained with the synthetic prosthesis, it would seem to be the more desirable procedure.

We are cognizant of the fact that our prostheses have been evaluated for periods of only up to 6 months following surgery but we feel that the results achieved, to date, with the rather crudely constructed grafts, justify this report.

Last, the incidence of the maintained patency of 75 per cent of the porous sponge Teflon prostheses, which was achieved in these canine studies, makes the use of similar Teflon prostheses as venous substitutes worthy of more detailed and extensive investigations. Such studies are currently under way in our laboratories.

SUMMARY

The experimental use of porous sponge Teflon protheses for superior vena caval replacement in canine animals has been presented. The results obtained, as reported herein, justify more extensive investigative amplification. If the results achieved in these studies are maintained in long-term evaluation studies, the clinical utilization of porous sponge Teflon as a superior vena caval prosthesis is indicated.

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OPERATION OF THE ROLLER PUMP FOR EXTRACORPOREAL CIRCULATION

Louis R. Head, M.D., Joop P. Coenen, M.D., Enrique Angola, M.D.,***
Cid Nogueira, M.D., David Mendelsohn, M.D., and
Earle B. Kay, M.D., Cleveland, Ohio

THE KAY-CROSS rotating disc oxygenator has now been used in over 250 operative procedures. It has proved itself to be a safe, simple, and efficient method of oxygenating blood. Over 150 cardiac centers where this oxygenator is employed, either clinically or experimentally, have substantiated this view. The development of convoluted discs increases the gas exchange surface by 40 per cent over the flat surface discs.[1]

Two different types of pumping systems have been used in conjunction with the Kay-Cross disc oxygenator in this hospital. Sigmamotor pumps were used initially and performed quite satisfactorily. However, laboratory studies on pump output, arterial line pressure, hemolysis, and perfusion led to the use of the Pemco type of De Bakey roller pumps. These pumps have been used in the last 100 patients with what is considered to be a marked improvement in perfusion management. The hemodynamic aspects of this pumping mechanism are reported in this paper, and the principles used in carrying out perfusion pumping are stated.

DESCRIPTION OF THE PEMCO ROLLER PUMP

The Pemco roller pump† in its present form is shown in Fig. 1. A system of rollers compress a rubber latex pump chamber over a 200 degree arc with a circumferential distance of 11 inches. No valves are used. A manual variable speed control ranges between 0 to 80 r.p.m. The distance of each roller from the pumping tract can be adjusted by a small screw which alters its center of rotation in relation to the roller arm and thus its distance from the tract. The roller system may be removed from the central pump shaft and systems containing two, three, and four rollers inserted into the pump. (Fig. 2). The rubber latex pump chamber may be varied in size for optimum stroke volume and flow rate.

From the St. Vincent Charity Hospital, Cleveland, Ohio.
Received for publication March 30, 1959.
*Present address: Stadionweg 281II, Amsterdam-Z, Netherlands.
**Present address: Ave. Honduras QTA. ISA. RU., Urb Las Acacias, Caracas, Venezuela.
†Manufactured by Pemco, Inc., 5663 Brecksville Rd., Cleveland 31, Ohio.

Tygon tubing is used to connect the pump chamber to the arterial cannula and to the oxygenator. A 3 foot length of 1/2 inch internal diameter tubing is used to the oxygenator; a 7 foot length of 3/8 inch internal diameter tubing to the arterial cannula. The arterial cannulae are 3 1/2 inches long. Each

Fig. 1.

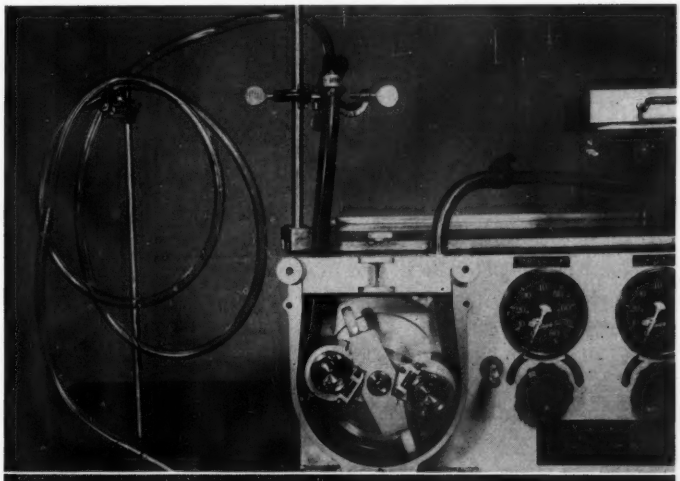


Fig. 2.

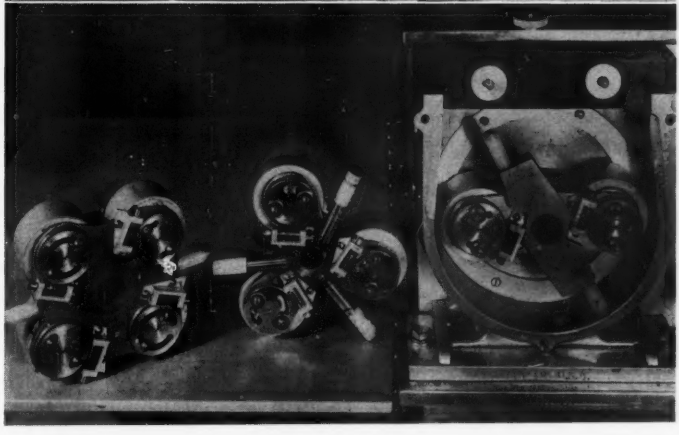


Fig. 1.—The Pemco roller pump with adjustable rollers, rubber latex pump chamber, 14 inch extension as depulsator, Tygon tubing, and the arterial cannula.

Fig. 2.—The two, three, and four roller systems.

consists of a stainless steel connector and Teflon tubing. The American wire gauge numbering system is used in referring to cannula size.

LABORATORY STUDIES

Each of the variable aspects of the pumping system, i. e., the roller setting, the number of rollers, the chamber size, and the cannula size was studied in

regard to the manner in which its alteration alone affected: (1) pump speed, flow rate, and arterial line pressure; (2) blood damage as indicated by hemolysis; and (3) perfusion of the patient as indicated by arterial pressure, flow, and vascular resistance.

METHODS

1. *Pump Calibration.*—Calibration of flow was done with blood at room temperature with the arterial cannula directed into a graduated cylinder 36 inches above the oxygenator reservoir. Flow per minute was determined at intervals of 10 r.p.m. Stroke volume was calculated as flow divided by revolutions per minute times the number of rollers in the system.

2. *Arterial Line Pressure.*—A No. 18 needle connected to a Statham transducer, which recorded on a Sanborn 150 recorder, was directed against the blood flow in the output end of the rubber latex pump chamber. This is the point of maximum pressure in the pump system. The resistance to flow at this point results from the Tygon connecting tubing, the cannula size and length, and the hydrostatic pressure equal to the distance between the blood reservoir and the tip of the arterial cannula in the graduated cylinder.

3. *Hemolysis.*—This is expressed as the change in the level of plasma hemoglobin with each passage of blood through the pump. Plasma hemoglobin was determined by a modification of the method of Hunter.[2] The recirculating system used to analyze the role of the pump in the production of hemolysis was the same in every way as that used during perfusion except that the rotating discs were left out of the oxygenator and no sump suction was fed into the system. In this way, the resulting hemolysis reflected only that produced by the pumping mechanism. Studies carried out with the discs in place and rotating indicate that there was no significant detectable damage to the blood from this source. In order to eliminate differences in red cell fragility and hematocrit, each series of tests were run from the same pool of blood. Fresh dog blood, anticoagulated with heparin, was used in 1,500 c.c. aliquots for each test run. Ten cubic centimeter samples of blood were taken from the arterial end of the oxygenator bottle at 15-minute intervals for runs of from 1 to 3 hours. The total hemolysis for each run was calculated as the difference in plasma hemoglobin in milligrams per cent, from the beginning of the run to the end. The hemolysis per blood passage was calculated from the flow rate, the circulating pool size, and expressed as milligrams per cent plasma hemoglobin.

4. *Perfusion Studies.*—Adult mongrel dogs, anesthetized with intravenous pentobarbital and anticoagulated with heparin, were perfused with the heart-lung machine. The arterial cannula was placed in the femoral artery. Venous blood was returned to the oxygenator through cannulae in each vena cava connected directly to the venous pump. Perfusion is controlled by maintaining a constant blood level in the oxygenator, and a normal venous pressure in the superior vena cava. This is correlated in conjunction with the appearance of the conjunctival vessels to maintain a constant blood volume and normal

blood pressure in the patient. In all instances, where the effects of the pump on perfusion were being determined, the heart was open and empty so that its action could not affect the perfusion. Aortic pressures were recorded from a catheter inserted into the abdominal aorta through the femoral artery. Pressure in the arterial line was taken in the tubing near the arterial cannula. The gradient across the arterial cannula was calculated as the difference between the mean arterial line pressure and the mean aortic pressure. In calculating the gradient across the animal's vascular system, the right auricular pressure was considered to be zero; the resistance of the vascular system being expressed as the ratio of the pressure gradient between the aorta and the right auricle to the calculated flow rate.

For clinical perfusions, the variable aspects of the pumping system must be adjusted to produce the flow rate selected for the individual. This and the size of the arterial cannula used are largely determined by the patient's weight and the caliber of the superficial femoral artery. Since both these factors are relatively fixed for each patient, the data on pump performance are presented from this standpoint, that is, to produce a given flow rate through a specific cannula.

RESULTS

Tables I, II, III, and IV present the data obtained from the studies on pump hemodynamics. These are self explanatory.

By tightly occluding the rollers, the amount of blood destruction per passage through the pump is increased (Table I). By adjusting the rollers to be nonocclusive, the large back flow through the pump decreases the stroke volume to the extent that excessively rapid pump speeds are necessary to produce a sufficient flow rate. With this setting, the affect of the resistance in the perfused subject markedly changes the calculated flow of the pump.

TABLE I. THE EFFECT OF ROLLER SETTING ON FLOW RATE, PUMP SPEED, ARTERIAL LINE PRESSURE FLOW REDUCTION WITH RESISTANCE, AND HEMOLYSIS USING THE TWO ROLLER PEMCO PUMP WITH A ¾ INCH I.D. CHAMBER AND A NO. 10 ARTERIAL CANNULA

ROLLER SETTING	NONOCCLUSIVE*	JUST NONOCCLUSIVE†	TIGHT OCCLUSIVE‡
Flow (c.c./min.)	2,000	2,000	2,000
Pump speed (r.p.m.)	50	23	21
Stroke volume (c.c.)	20	42	48
Arterial line pressure (mm. Hg)	410/240	400/200	420/180
Pulse pressure (mm. Hg)	170	200	240
Decrease in flow at 36 in. hydrostatic pressure	200	100	0
Hemolysis in mg. % per passage	0.045	0.054	0.090

*Nonocclusive setting = 300 c.c./min. back flow at 36 in. hydrostatic pressure.
†Just nonocclusive setting = no back flow at 36 in. hydrostatic pressure.
‡Tight occlusive setting = no back flow at any arterial line pressure.

Therefore, the just nonocclusive setting, by compromising between these two settings, decreases hemolysis and permits only a small back flow. This is the roller setting used in all of the pump studies. Because of this just nonocclusive roller setting, the pump must be calibrated against a simulated resistance, and there is a loss in stroke volume with an increase in the arterial line pressure (Table II).

TABLE II. THE EFFECT OF PUMP SPEED AND CANNULA SIZE ON STROKE VOLUME WITH THE JUST NONOCCLUSIVE SETTING OF THE ROLLERS

R.P.M.	STROKE VOLUME	
	CANNULA NO. 6 (C.C.)	CANNULA NO. 10 (C.C.)
10	66	39
20	53	32
30	55	31
40	53	31
50	49	30
60	48	30
70	47	29
80	47	28

The addition of rollers to the pumping system (Table III) shortens the effective pumping tract and decreases the stroke volume without decreasing the distance that each roller must travel during the pump cycle. Therefore, hemolysis is markedly increased. The addition of four rollers to the pumping system extends the speed range of the pump from 160 to 320 strokes per minute, without significantly increasing the flow rate. When the stroke volume is reduced by adding rollers, a given flow can be pumped with a lower pump speed in revolutions per minute than when the same flow rate is pumped using fewer rollers.

TABLE III. THE EFFECT OF ROLLER NUMBER AND CHAMBER SIZE ON ARTERIAL LINE PRESSURE, PUMP SPEED, STROKE VOLUME, PULSE PRESSURE, AND HEMOLYSIS WHEN PUMPING 2,000 C.C./MINUTE THROUGH A NO. 10 ARTERIAL CANNULA WITH THE PEMCO ROLLER PUMP SET TO BE JUST NONOCCLUSIVE FOR BLOOD

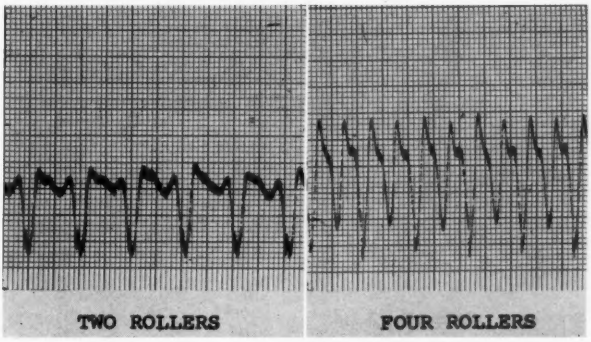
Roller number	2	4	2	2
Chamber size in inches I.D.	½	½	¾	⅜
Flow (c.c./min.)	2,000	2,000	2,000	2,000
Pump speed (r.p.m.)	33	41	24	76
Stroke volume (c.c.)	30	12	42	13
Arterial line pressure (mm. Hg)	390/190	410/150	400/200	420/200
Pulse pressure (mm. Hg)	200	260	200	220
Hemolysis in mg. % per passage	0.40	1.0	0.06	0.50

The configuration of the pulse pressure curve within the pump is influenced by the length of the pump tract (Fig. 3, *A*). By lengthening the tract, the ejection of the stroke volume is prolonged and the maximum pressure and flow sustained over a larger portion of the pump cycle. This is in contrast to the action of the heart wherein the major portion of the stroke volume is ejected in one tenth of the cycle.

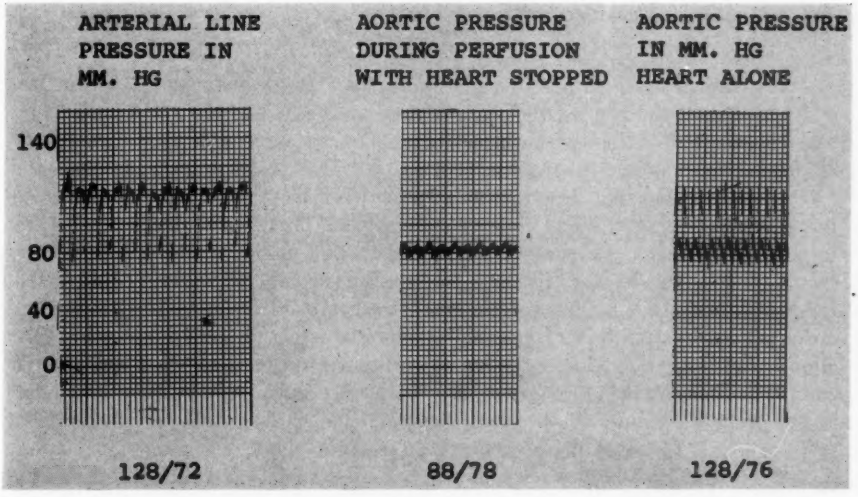
Decreasing the size of the pump chamber decreases the stroke volume, but necessitates an increase in pump speed from 24 to 76 r.p.m. in order to maintain the flow rate (Table III). This limits the flow range at which the smaller stroke volumes can be used. By enlarging the chamber, the flow range of the pump is extended. The hemolysis produced by the 3/8 inch pump chamber is significantly less than that seen when an equal reduction in stroke volume is produced by the four roller system (Table III).

Enlarging the internal diameter of the arterial cannula, either by using two vessels of the same size or placing it in a single larger vessel, significantly

lowers the arterial line pressure at which a given flow is reached (Table IV). This lowering of pressure and, thus, pressure gradient across the arterial cannula, is not, however, associated with a significant decrease in the level of hemolysis.



A.



B.

Fig. 3.—*A*, Arterial line pulse pressure curves using the Pemco two and four roller systems.

B, Arterial line and aortic pressure curves, during perfusion with a two roller Pemco pump, ⅜ inch I.D. chamber and No. 10 cannula, compared to aortic pressure curve with the heart alone.

The significant factor responsible for hemolysis from the pump is the compression of the pump chamber by occlusive rollers (Tables I, III, IV, and V). Stroke volume affects hemolysis only as it influences pump speed. The pressure gradient across the arterial cannula does play some role in blood damage, but quantitatively this is small. The absolute level of the arterial line pressure has no influence on hemolysis (Table V).

TABLE IV. THE EFFECT OF CANNULA SIZE AND NUMBER ON PUMP SPEED, STROKE VOLUME, ARTERIAL LINE PRESSURE, PULSE PRESSURE, AND HEMOLYSIS WHEN PUMPING 2,500 C.C./MINUTE WITH A ⅝ INCH PUMP CHAMBER OF A TWO ROLLER PEMCO PUMP

Cannula size	No. 10	No. 6	No. 10 (2)
Flow (c.c./min.)	2,500	2,500	2,500
Pump speed (r.p.m.)	39	31	22
Stroke volume (c.c.)	32	40	57
Arterial line pressure (mm. Hg)	600/390	230/30	170/40
Pulse pressure (mm. Hg)	210	200	130
Hemolysis in mg. % per blood passage	0.4	0.3	0.4

TABLE V. COMPARISON OF PUMP SPEED AND ARTERIAL LINE PRESSURE IN THE PRODUCTION OF BLOOD DAMAGE EXPRESSED AS PLASMA HEMOGLOBIN IN MG. % RESULTING FROM THE CIRCULATION OF BLOOD THROUGH THE PEMCO ROLLER PUMP SET TO BE JUST NONOCCLUSIVE FOR BLOOD

Roller number	2	2	4	4	2	2
Pump speed	20	60	20	60	60	60
Arterial line pressure	—	—	—	—	272/112	640/440
Cannula size	None	None	None	None	6	10
Priming volume	1,500	1,500	1,500	1,500	1,500	1,500
Length of run (min.)	60	60	60	60	60	60
Flow per minute (c.c.)	1,675	4,440	1,190	3,366	3,950	2,360
Total blood passage	67	172	42	130	130	81
Total hemolysis	32	66	33	141	37	33
Total hemolysis per blood passage in mg. %	0.48	0.4	0.79	1.0	0.3	0.4

The effects of the roller type of pump on the hemodynamics within the blood stream of the perfused subject is presented in Tables VI, VII, and VIII.

The level of aortic pressure during perfusion is related to the flow rate as long as the quantity of blood in the perfused subject remains constant (Table VI). However, the relationship between flow and aortic pressure is not a linear one, the higher flow rates being associated with a decreased effect on blood pressure. In the normal individual, this, in all probability, is a result of the moderator reflexes originating from the carotid sinus and tending to maintain a constant arterial pressure over a wide range of cardiac outputs.[3] In situations encountered during open heart surgery, where abnormally large quantities of blood are returned to the heart chambers via the coronary sinus, bronchial arteries, or incompetent aortic valve, this relationship does not hold. Under these circumstances, the rate of flow has little effect upon the level of aortic pressure beyond a certain point.

TABLE VI. THE EFFECT OF FLOW RATE ON PULSE PRESSURE, MEAN AORTIC PRESSURE, AND VASCULAR RESISTANCE IN A 16 KG. DOG USING A 3 ROLLER PUMP WITH A ½ INCH CHAMBER AND A NO. 10 CANNULA

Cubic centimeters per kilogram	50	75	90	170
Cubic centimeters per minute	820	1,230	1,470	2,800
Pump speed (r.p.m.)	15	25	32	60
Stroke volume (c.c.)	18	16	15	15
Mean aortic pressure	60	70	80	100
Aortic pulse pressure	8	8	8	4
Vascular resistance (mean pressure/flow)	0.07	0.06	0.05	0.04

The pulsations within the aorta are considerably dampened when compared to those in the pump lines and in the blood stream when perfusion is carried

on by the heart action (Table VII). This depulsated flow produced by the roller type of pump results mainly from the continuous nature of its pumping action. The duration of time during the pump cycle that the rollers are not functioning is short. This brief period is that in which the pressure fluctuations occur (Fig. 3, *B*). The width of the pulse pressure in the aorta is also affected by the stroke volume and the pressure gradient across the arterial cannula (Tables VII, VIII). Increasing the stroke volume from 17 to 70 c.c. doubles the pulse pressure in the aorta.

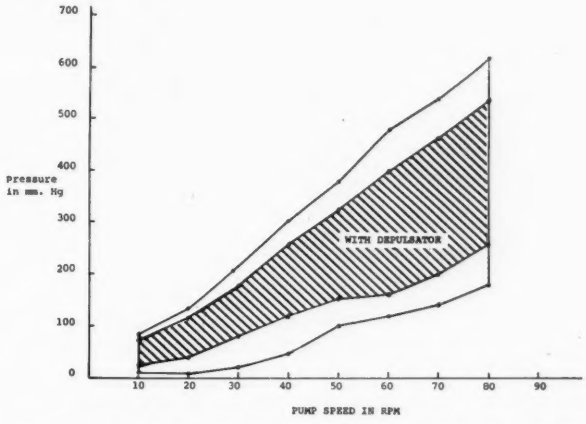


Fig. 4.—Arterial line pressure with and without a 14 inch depulsator.

TABLE VII. THE EFFECT OF STROKE VOLUME ON PUMP SPEED, ARTERIAL LINE PRESSURE, AORTIC PRESSURE, AND THE AORTIC PULSE PRESSURE WHEN FLOWING 700 C.C. PER MINUTE THROUGH A NO. 9 CANNULA USING THE ROLLER PUMP AND THE SIGMAMOTOR PUMP

Pump type and chamber	2 roller 3/8 in.	2 roller 3/4 in.	Sigmamotor 3/8 in.
Flow (c.c./min.)	700	700	700
Pump speed (r.p.m.)	20	5	114
Stroke volume	17	70	6
Arterial line pressure (mm. Hg)	168/100	168/108	168/64
Aortic pressure (mm. Hg)	120/108	128/104	72/60
Aortic pulse pressure (mm. Hg)	12	24	12

TABLE VIII. THE EFFECT OF PRESSURE GRADIENT ON FLOW RATE, PUMP SPEED, STROKE VOLUME, AORTIC PRESSURE, AND AORTIC PULSE PRESSURE USING A 2 ROLLER PEMCO PUMP WITH A ⅜ INCH CHAMBER AND NO. 9 AND NO. 12 ARTERIAL CANNULAE

Cannula number	9	12
Flow (c.c./min.)	1,050	1,050
Pump speed (r.p.m.)	30	40
Stroke volume (c.c.)	18	13
Arterial line pressure (mm. Hg)	208/80	780/380
Aortic pressure (mm. Hg)	72/60	108/88
Aortic pulse pressure (mm. Hg)	12	20

The pressure fluctuations within the pump tubing reflect the differences in elasticity between it and the blood vessels. Here in the pump tubing the pulse pressure increases as the arterial line pressure rises and the tubing becomes inelastic. The elasticity in the pump assemble can be increased by extending the rubber latex pump chamber from 7 to 14 inches beyond the pump output head (see Fig. 1). This decreases the line pulsations by lowering the systolic pressure and raising the diastolic. There is no change in flow rate, mean line pressure, or hemolysis resulting from this rubber depulsator (Fig. 4).

DISCUSSION

From these studies it is apparent that the Pemco roller pump is capable of producing a pump flow with a variety of characteristics. The range of stroke volume may be adjusted between 3 and 70 c.c. The number of strokes per minute can be controlled between 0 to 320. The rollers can be adjusted so as to control the back flow through the pump. The pulsatile characteristics of the flow may be controlled by adjusting the elasticity of the pump tubing and the level of pressure gradient across the arterial cannula. This range of adaptability makes the pump suitable for incorporation into any system of extracorporeal circulation. It allows the characteristic of the pump flow to be regulated, within limits, to incorporate a variety of perfusion concepts.

The method of perfusion control developed for the Kay-Cross disc oxygenator, as used during the past 250 operative cases, has been outlined under methods of perfusion. The following method of pump assembly has been found to facilitate the operation of this perfusion. It has lowered the level of hemolysis resulting from cardiac bypass with the pump oxygenator. It has not been associated with any detectable disadvantages.

1. *Hemolysis.*—Three considerations have been employed to reduce blood damage. The rollers are not tightened to completely occlude the pump chamber. The three and four roller systems are not used. The speed of the pump during perfusion has been reduced by adjusting the stroke volume of the pump.

2. *Flow Characteristics.*—A flow as nonpulsatile as possible is used both through the patient and the extracorporeal system. This is done by not using large stroke volumes or high pressure gradients. Small stroke volumes are also avoided, since the associated increase in pump speed raises hemolysis. Rubber latex depulsator tubing is used on both the arterial and venous pumps as a means of further decreasing the pulsatile characteristics of the flow occurring from the use of intermediate stroke volumes.

3. *Pump Speed.*—All patients are perfused in the same range of pump speed, between 30 to 60 r.p.m. This is done by correlating the size of the pump chamber with the arterial cannula to be used so that the stroke volume of the pump will be such that a pump speed of 40 r.p.m. will produce a flow rate of 75 c.c. per kilogram per minute. This places what may be considered the patient's resting cardiac output in the center of the pump speed range at a pump speed equal to the normal heart rate, 80 beats per minute.

This maneuver calibrates the pump speed range to fit the patient being perfused. It allows flow rates up to 170 c.c. per kilogram per minute to be administered to the patient by adjusting the pump speed.

4. *Pressure Gradient.*—A single arterial cannula placed into the femoral artery through the superficial femoral artery is used in all instances. The safety of the pump connections has been considered to be the major factor in adjusting the pressure gradient across the arterial cannula. If the maximum pressure in the arterial line is below 500 mm. Hg, it is believed to be in a safe range if the connections are properly fixed. There is no major advantage to be gained from the standpoint of hemolysis or flow rate by maintaining the lowest possible pressure gradient between the pump and the patient.

With the pump speed calibrated to fit the patient, the pump speed approximately that of the heart rate, and a nonpulsatile flow, the perfusion is smoothly controlled. A steady flow from the venous cannulae, a smooth surface of the oxygenator blood level, and a small pulse pressure in the ascending aorta[6] are all considered to be advantageous characteristics obtained from this type of pump operation.

A comparison of similar groups of patients perfused for the repair of ventricular septal defects, with the Sigmamotor pumps and with the Pemco type of roller pumps, indicates that the total level of hemolysis has decreased from an average of 88 mg. per cent to 44 mg. per cent.

It should be kept in mind that this type of perfusion does not exactly reproduce the pumping action of the heart.[4, 5] Reproduction of the cardiac action during perfusion, although more physiologic from a theoretical point of view, would present serious disadvantages when used with an extracorporeal oxygenating system and method of perfusion dependent to some extent on nonpulsatile flow. As there have been no detrimental effects of this type of perfusion on the patient, it is felt that there is little to be gained in modifying the pump to be less continuous. The unavoidable existence of a pressure gradient between the pump and the patient, the advantages of a nonpulsatile type of flow from the standpoint of oxygenator function, and the desirability of eliminating large pressure fluctuations in the aorta while the left auricle is open,[6] all contribute to the suitability of a continuous flow pump in this type of extracorporeal circulation.

SUMMARY

Hemodynamic studies on the Pemco type of De Bakey roller pump indicates that it can be adjusted to operate occlusively or nonocclusively. The speed of the rollers can be increased so as to produce up to 320 strokes of the rollers per minute. The stroke volume can be varied between 3 to 70 c.c. Elongation of the pump chamber increases the elasticity in the pump tubing and acts as a depulsator.

The major factor responsible for hemolysis due to the pump is the compression of the pump chamber by occlusive rollers and the number of strokes per minute to produce a given flow. The multiple roller system

containing three and four rollers, although extending the speed range of the pump, significantly increases hemolysis. The level of the pressure gradient and the size of the stroke volume have little quantitative role in blood damage.

A method of pump operation wherein the pump is calibrated to fit the cardiac output range of the patient, the pump speed approximately that of the heart rate, and the flow of a nonpulsatile quality, has been found to produce little hemolysis, to be easily controlled, and to work well with the Kay-Cross disc oxygenator.

This type of perfusion has been used in over 100 operative cases. It has lowered hemolysis without changing the nonpulsatile characteristics of the flow.

We wish to express our appreciation to Dr. Matthew Levy, Director, Department of Research, St. Vincent Charity Hospital; Andre Lux, Clinton Blazek, and Janet Osborn for technical assistance, and to Harry Macko, medical technologist, for chemical analysis of plasma hemoglobin.

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VACUUM REGULATOR FOR CARDIOTOMY RETURN AND CHEST DRAINAGE SYSTEMS

Nazih Zuhdi, M.D., Garman Kimmell, John Carey, M.D.,** and Allen Greer, M.D.,*** Oklahoma City, Okla.*

THE USUAL method of regulating the amount of suction applied to the cardiotomy return system during open heart surgery or to the pleural cavity following cardiopulmonary surgery is governed by the submergence of an open end glass tube below the water or mercury level in a container subjected to a source of suction.[1, 2] In order to increase the amount of suction, the depth of submergence is increased, a plastic tube at the end of the air vent is occluded or several bottles are placed in series. Often the resultant vacuum is not known. Encumbrance of the setup, breakage of the containers or air vent tube, spillage of water and mercury, and leaks around the fittings or the corks are additional factors which justify the construction of a small, inexpensive, simple, durable, and accurate vacuum regulator.†

DESCRIPTION AND PERFORMANCE OF VACUUM REGULATOR

Fig. 1 shows the external appearance of the regulator and Fig. 2 the details of its construction. It is about 9 cm. in diameter and 13 cm. in height. It has a central knob that indicates, in centimeters of mercury, the amount of vacuum that will be held. The knob applies a force through an Inconel‡ spring to a seat disc which reacts to the vacuum and regulates the amount of room air admitted into the line connecting the source of suction to the container. The resultant vacuum is maintained close to the setting of the knob (Fig. 5). The vacuum range of the regulator is a function of the inner spring. At present, the range is from 0 to 10 cm. of mercury, marked in centimeter graduations. Fig. 3 demonstrates the position of the regulator in the cardiotomy return system during open heart surgery and Fig. 4 in pleural drainage. Fig. 5 depicts the pressure flow characteristics of the vacuum regulator.

From the Department of Surgery, Mercy Hospital, Oklahoma City, Okla.
Received for publication April 1, 1959.
*Present address: 430 N. W. 12, Oklahoma City, Okla.
**Instructor in Surgery, Oklahoma University Medical School, Oklahoma City, Okla.
***Assistant Professor of Surgery, Oklahoma University Medical School, Oklahoma City, Okla.
†Designed by Garman Kimmell. Manufactured by Kimray Inc., 52 N.W. 42, Oklahoma City, Okla.
‡This low-stressed corrosion resistant spring should not require replacement or recalibration.

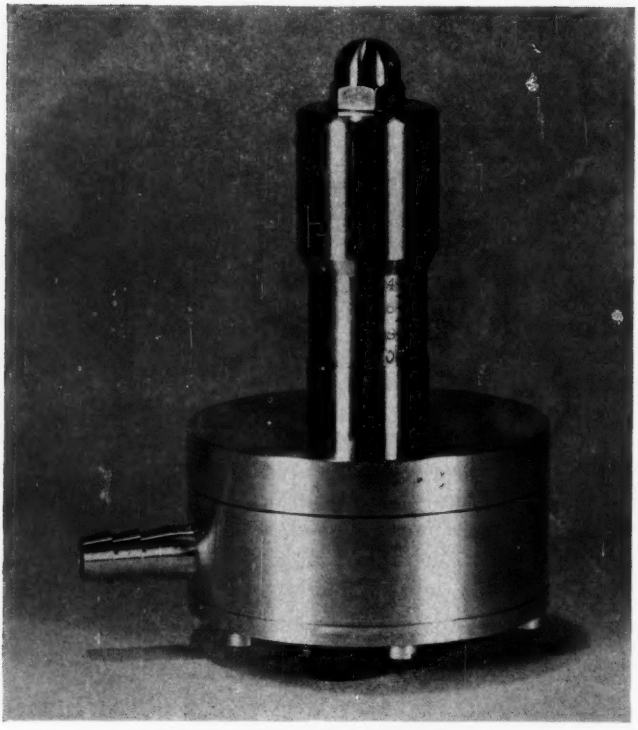

Fig. 1.—External appearance of vacuum regulator. It is compact, simple, and accurate. Calibrations are in centimeters of mercury.

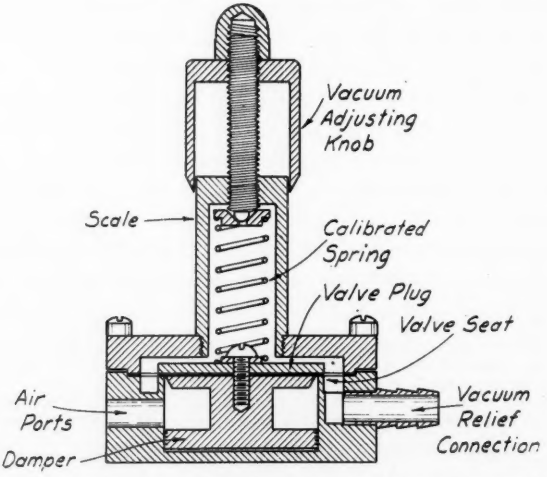

Fig. 2.—Details of the construction of the apparatus. The Inconel spring does not require recalibration.

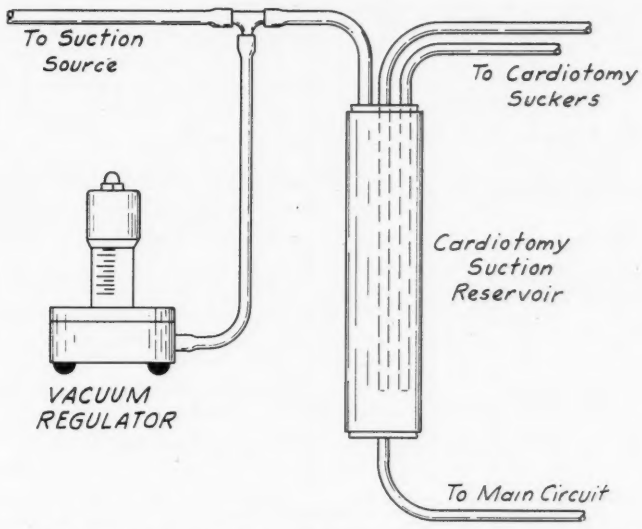

Fig. 3.—Position of vacuum regulator in cardiotomy return. Readiness and simplicity are major features.

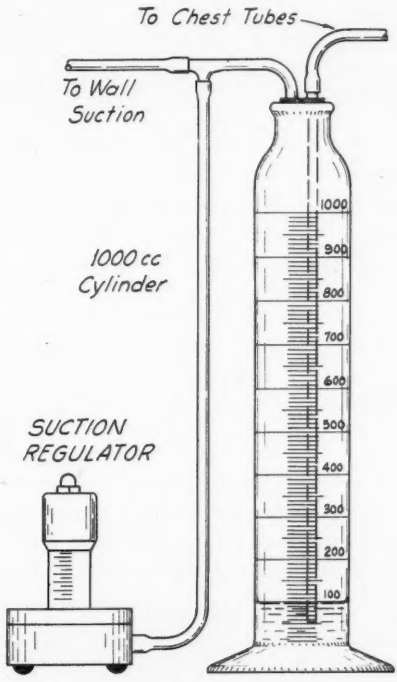

Fig. 4.—Position of regulator in pleural drainage. Accurate measurements of blood loss are feasible and vacuum pressure changes instantly accomplished.

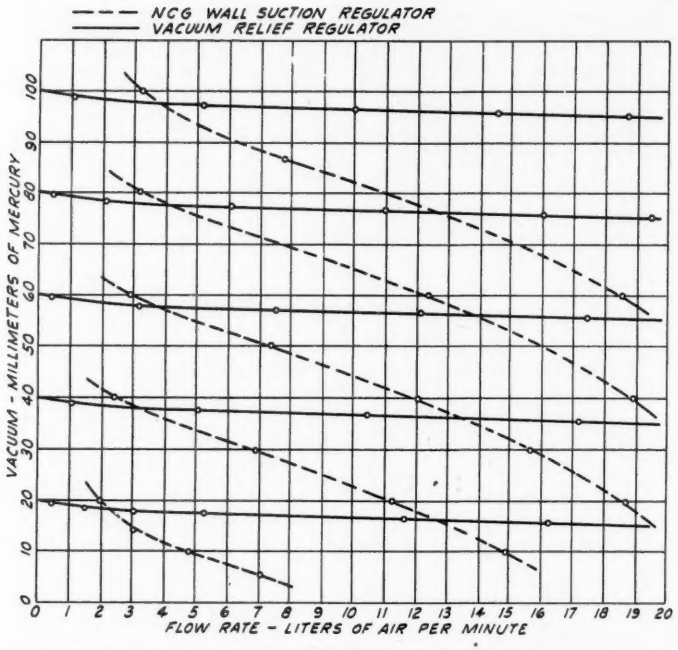
NCG WALL SUCTION REGULATOR
VACUUM RELIEF REGULATOR
VACUUM - MILLIMETERS OF MERCURY
0 10 20 30 40 50 60 70 80 90 100
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20
FLOW RATE - LITERS OF AIR PER MINUTE

Fig. 5.—Flow-pressure characteristics of vacuum regulator up to 20 L. per minute are shown together with the control characteristics of wall suction installation (NCG 238-50V). Vacuum pressure changes of only 0.5 cm. Hg occur with air flow rates from close to 0 to 20 L. per minute.

CONCLUSION

A vacuum regulator is described for the cardiotomy return and pleural cavity drainage system. It is small in size, dependable, unbreakable, and the vacuum applied is known and maintained at all times.

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THE DESIGN OF MONITORING DEVICES FOR USE WITH A PUMP-OXYGENATOR

Vernon Montgomery, M.D., Ph.D., Bruce C. Paton, M.R.C.P., F.R.C.S., Jack Lucero, and Henry Swan, M.D., D.Sc., Denver, Colo.*

THERE HAS been much discussion, since the use of extracorporeal circulation became widespread, about the principles of perfusion that will give optimal results. In this hospital the problem has been approached, not on the basis of high or low flows, but by attempting to maintain the blood gas relationships within normal limits. It is felt that if sufficient oxygenated blood is provided by a pump-oxygenator to maintain a reasonable perfusion pressure, and if the quantity and rate of delivery of oxygenated blood results in a normal arterial-venous oxygen difference, then adequate perfusion has been attained. The actual amount of blood perfused in cubic centimeters per kilogram per minute varies from patient to patient, but as judged by the oxygen consumption of the patient and the adequacy with which this consumption is met, the perfusion is adequate.

In order that perfusions may be conducted according to these principles, monitoring devices have been introduced into the extracorporeal circuit. It is necessary to monitor simultaneously and continuously arterial and venous oxygen tensions and arterial pH in the machine. In addition, in the patient, mean arterial and venous pressures, electroencephelogram, temperature, and electrocardiogram are also measured. The arterial oxygen tension and the pump speed determine the disc speed required. At a given pump speed, the greater the disc speed the greater the degree of oxygenation obtained. The pump speed is determined by the perfusion pressure, the venous oxygen tension, and the electroencephalogram. A small arterial-venous oxygen difference is taken to mean overperfusion and too large an arterial-venous oxygen difference indicates underperfusion.

Control of carbon dioxide tension in arterial blood as estimated by pH is acomplished by altering the per cent carbon dioxide in the gas "respired" in the oxygenator. Two gas mixtures of oxygen and carbon dioxide containing 2 per cent and 4 per cent, respectively, are used. A total flow of about

From the Halsted Laboratory of Experimental Surgery and the Department of Surgery, University of Colorado School of Medicine, Denver, Colo.

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12 L. per minute is satisfactory and, by varying the proportions of each mixture, the percentage of carbon dioxide can be altered and, thus, the pH maintained within normal limits.

The pH is measured by a Beckman Zeromatic meter which reads continuously and has a self-correcting device actuated every second. It is accurate to within plus or minus 0.05 pH and is thoroughly reliable and simple to operate. At the start of each perfusion, a sample of pump blood is taken off and its pH read on a Beckman Model G meter and the calibration of the Zeromatic meter is thereby checked.

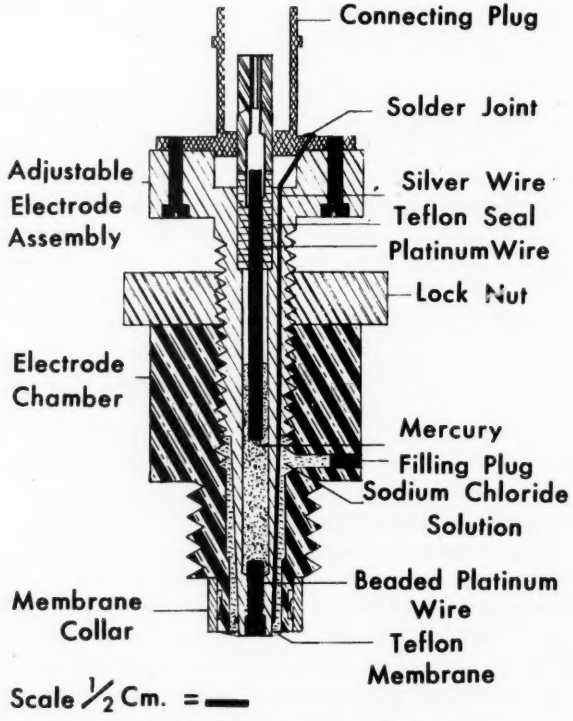

Fig. 1.—Membrane-covered polarograph.

The need for continuous monitoring of pO_2 necessitated the design of special electrodes (Fig. 1). The electrodes used are modifications of the Clark[1] polarograph. The electrode unit is in two parts; an inner core containing the electrodes and an outer chamber enclosing the electrode assembly. The platinum electrode lies in the center of the core and is divided at its middle. The separated ends dip into a sealed pool of mercury and in this way electrical contact is maintained while the differences between the coefficients of expansion of Teflon and platinum are eliminated. The silver

electrode lies along the outside of the core and its tip encircles the Teflon rod. A Teflon membrane, 0.002 inch thick and 0.5 inch in diameter, is held in place over the end of the electrode chamber by a removable collar.

The use of Teflon rather than other plastics permits the electrode to be autoclaved. This is done with the electrode disassembled. To reassemble the unit, the membrane is first secured in position and the electrode chamber filled with normal saline solution. During filling, a small screw plug is removed from the side of the chamber through which air bubbles may escape while the electrode assembly is being screwed into position. When the tip of the platinum electrode is sufficiently advanced beyond the end of the chamber to make the membrane tense and slightly tented, the lock nut on the assembly is tightened. No further change can then occur in the position of the electrode tip.

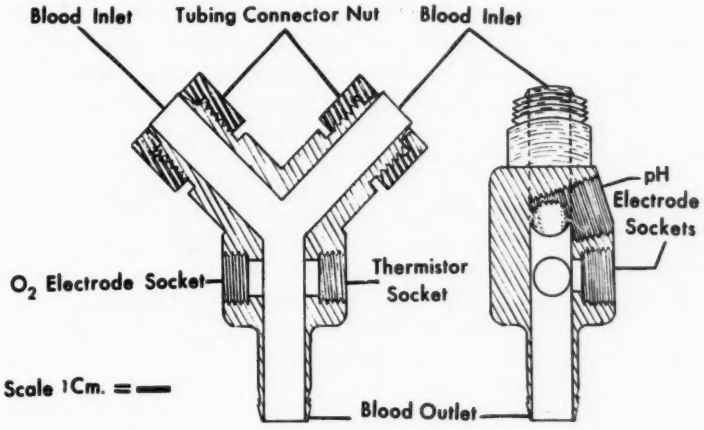

Fig. 2.—Multi-electrode adaptor.

The polarographs are connected with circuit control units so calibrated as to register oxygen tensions directly. The circuit used in this unit is similar to that described by Severinghaus and Bradley,[2] but has been modified to deliver a potential difference of 0.7 volt across the electrodes.

Under both experimental and clinical conditions, these polarographs have been found to react almost instantly to changes in oxygen tension, but are not so sensitive as to be difficult to control. They are not affected by changes in temperature or pressure in the blood, but slight changes occur in the readings with variations in blood flow rates.

In order to introduce these electrodes into the circuit, special adaptors have been designed to house them (Fig. 2). At the venous end, the adaptor consists of a simple tube with a side hole into which the polarograph is screwed. This adaptor also has another attachment to which a short-circuiting tube from the arterial end of the circuit for use during the period of equilibration before perfusion may be attached. At the arterial end the adaptor is more complicated and is located between the outflow tube from the oxygenating

cylinder and the arterial pump. It consists of a Y junction rejoining the blood flows from the two oxygenating cylinders and in the sides of which are screw plugs containing a polarograph, thermistor, and two pH meter electrodes. The dimensions of the adaptor are given in Table I.

TABLE I

	VENOUS ADAPTOR	ARTERIAL ADAPTOR
Length	5 15/16 in.	3 7/8 in.
External diameter	1 9/16 in.	1 in.
Internal diameter	5/8 in.	1/2 in.

SUMMARY

The principles of perfusion used at the University of Colorado and the consequent need for continuous monitoring devices have been described. A plea is made for attempting to achieve "physiologic" perfusion.

Details are given of an autoclavable polarograph and multi-electrode adaptor for the continuous monitoring of extracorporeal perfusion.

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THE PREVENTION OF HEART BLOCK IN THE SURGICAL REPAIR OF INTERVENTRICULAR SEPTAL DEFECT

Howard D. Sirak, M.D., Charles V. Meckstroth, M.D., Joseph M. Ryan, M.D., Leon G. Claassen, M.D., and Don M. Hosier, M.D., Columbus, Ohio

HEART BLOCK is reported to be an important cause of morbidity and mortality following surgical correction of interventricular septal defect.[2, 3] If this complication were eliminated, operative risk in most cases would then be minimal and surgery could justifiably be recommended for less seriously ill patients. In this way, many individuals could enjoy full activity and normal growth and development which they otherwise would be denied.

Current figures, if they were available, would undoubtedly show that this complication is encountered less frequently now because of the improvements in perfusion methods and operative technique. However, the incidence is still significant.[3] When heart block occurs, its management is greatly facilitated by the myocardial electrode and electronic pacemaker,[9] but in those patients who have persistence of complete atrioventricular block, cardiac function may be seriously compromised. They are prone also to sudden death.

Heart block has not occurred in our series of 53 cases of interventricular septal defect. The surgical technique to be described seems to have eliminated the problem of heart block while providing a reliable method for obtaining complete closure of each type of defect.

SELECTION OF PATIENTS

Initially, only those patients with pulmonary hypertension were accepted for operation. As our experience increased and operative risk was shown to be minimal in the less severe cases, the criteria for selection were liberalized. At present, in addition to the pulmonary hypertensive group, those patients who have evidence of large left-to-right shunts are also advised to have operation. The minimal operative mortality and morbidity in these cases has justified this decision.

PATCH DESIGN

A satisfactory technique should provide secure closure of the septal defect irrespective of its size and location or the characteristics of the surrounding

From the Cardiovascular Service and the Departments of Cardiology, Anesthesiology and Pediatric Cardiology, The Ohio State University Health Center and the Children's Hospital, Columbus, Ohio.

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septal tissue. It must consistently avoid injury to the conduction system of the heart. Because an actual tissue deficit is present, a substitute plastic patch material would seem desirable. This is particularly true in the larger defect or in those in which the septal rim is thin. If no patch is employed, tension on the sutures, with ensuing edema and punctate hemorrhage, increases the likelihood of conduction disturbances. Also, disruption of the repair under the stresses of heart contraction might occur.

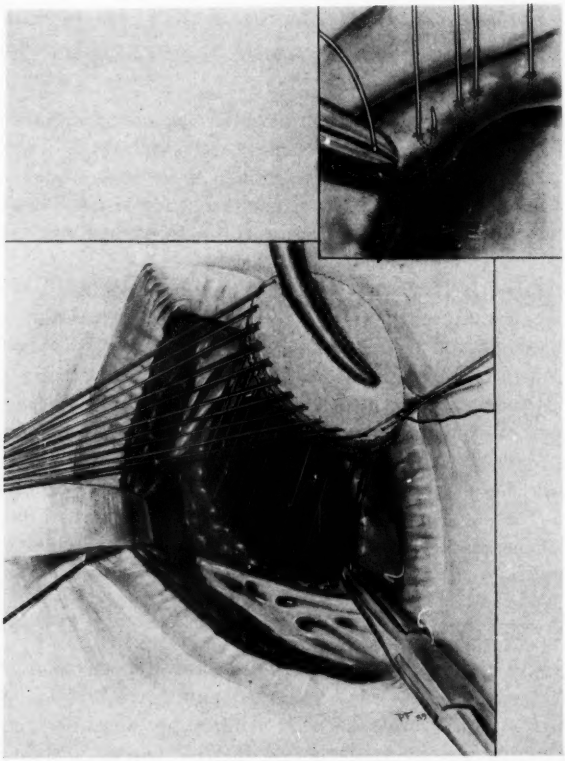

Fig. 1.—This drawing shows the two-component patch being used to close a large defect. The overlapping of the sutures is shown. The noncompressed polyvinyl sponge lies against the septum; the Teflon felt faces the cavity of the right ventricle. The *inset* illustrates the method of placing the Mersilene sutures in the rim of the defect so that they lie parallel to the course of the conduction fibers. Note the slight overlapping of the mattress sutures.

The danger of incorporating the conduction fibers in the sutures being placed in the posterior rim of the defect has been adequately emphasized.[1, 8, 10] However, because these pathways lie just beneath the endocardium,[4] they may easily be traumatized if they are pressed against a hard, semirigid patch material when the sutures are tied down. This is particularly true when the white fibrous tissue is absent from the posterior rim, and the edge of the patch must lie against the muscle containing these fibers. The possible role of injury from patch compression has not been sufficiently stressed.

The plastic cloth patch that has proved to be most satisfactory consists of two components. They are: polyvinyl (Ivalon) sponge and Teflon felt.* The Ivalon sponge is 2 mm. thick and is not compressed. It provides the desired cushion effect and applies itself evenly to the contours of the septal rim. The Teflon felt is 2 mm. thick and faces the cavity of the right ventricle (Fig. 1). It holds sutures well; it is flexible and provides the needed strength for the patch. Thus, a firm repair of the larger defects is obtained without having to

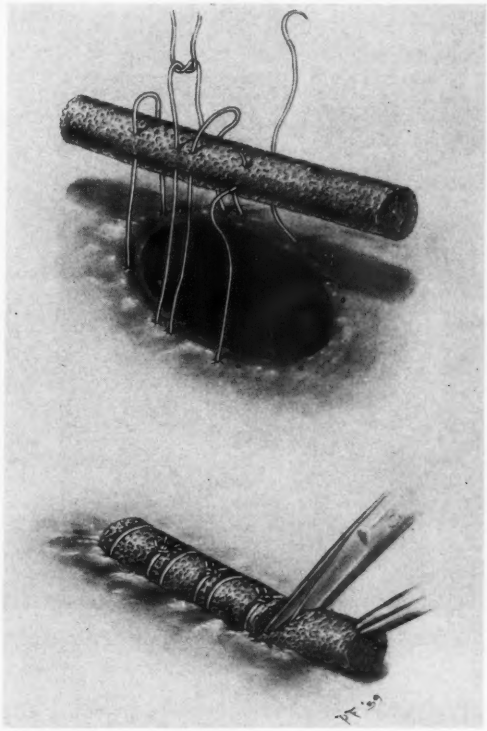

Fig. 2.—This drawing illustrates the use of a noncompressed cigaret-shaped polyvinyl sponge to close smaller defects with firm fibrous rims.

tighten the sutures excessively and risk crushing the septum. Usually two or three sizes are prepared prior to starting the bypass. They are held in readiness in a tonsil hemostat.

For defects around 1 cm. in size with strong fibrous rims, an alternate patch material has been used. This consists of a cigarette-shaped piece of noncompressed polyvinyl sponge (Fig. 2). It is prepared with cork borers in two sizes, 1/4 inch and 3/8 inch in diameter.

*Available from Mr. Norman C. Jeckel, United States Catheter and Instrument Corp., Glens Falls, N. Y.

As an additional precautionary measure, Mersilene* sutures have been employed routinely for all septal suturing during the past year because of their smoother surface and low tissue reactivity. These are double-armed with sturdy small needles excellently suited for work in this crowded area.

OPERATION

A mechanical heart-lung apparatus of the Kay-Cross type was employed in all cases. Potassium citrate arrest was used routinely. A vertical sternotomy is now used exclusively except in those patients with an associated patent ductus arteriosus.

Fig. 3.—This photograph demonstrates the hemostat holder which keeps each successive suture in sequence, thereby preventing entanglement. Two are used, one for each side of the septal rim.

Technique.—

1. *Large defects over 1 cm. in diameter or those with thin rims:* A series of 3-0 atraumatic Mersilene sutures are placed mattress-fashion around and parallel to the rim of the defect so that each suture slightly overlaps the preceding suture (Fig. 1). In this way, the course of the needle through the tissue and the imbedded suture itself always lies parallel to the course of the conduction fibers. The first suture is placed in the anterosuperior angle of the defect. After the suture is placed in the septum, the two ends are separately passed about 3 mm. from the edge through both layers of the patch being held by the assistant in a tonsil hemostat. A patch which is large enough to allow a 2 to 3 mm. overlap at the edges is chosen. The suture ends are held together in a

*Mersilene sutures are Du Pont's Dacron polyester fiber. Supplied in 24 inch lengths with 2 RB-1 needles by Mr. C. W. Norman, Ethicon, Inc., Somerville, N. J.

hemostat, the needles are cut off, and the hemostat is placed in a special hemostat-holding bracket located at each side of the operative field (Fig. 3). These brackets keep the hemostats in order and prevent later entanglement and confusion while the patch is being tied in place. The subsequent sutures are placed counterclockwise proceeding along the posterior rim of the margin of the defect toward the postero-inferior angle. Full-thickness sutures are placed in the white fibrous tissue if it appears strong. If the posterior rim of the defect appears thin, the sutures are placed in the adjacent muscle. Here, a more superficial bite is taken, but a broader area of tissue is incorporated in the mattress suture. At the postero-inferior angle, the suture is placed as close to the edge of the septum as possible. The remainder of the sutures are then

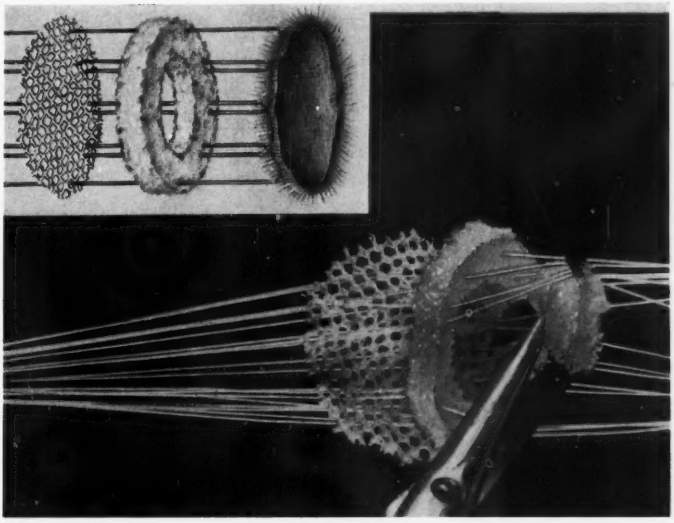

Fig. 4.—This illustration shows the modified septal patch for cases with severe pulmonary hypertension due to high vascular resistance. The cushion effect of the polyvinyl sponge is retained and the mesh permits gradual obliteration of the shunt.

placed along the anterior rim, proceeding from the postero-inferior angle to the starting point. This part of the septum is usually muscular and is away from the conduction fibers. Deeper sutures should be taken to obtain secure closure. The patch is then put in place with the tonsil hemostat, and the sutures are tied down in reverse order of their insertion. Just before the last one is tied, the left ventricle is compressed with the hand to express entrapped air bubbles. Each suture is cut about 1 to 2 mm. from the knot.

In the presence of high pulmonary vascular resistance, the patch is modified to permit gradual closure; in such patients, a Teflon cloth mesh* is substituted for the Teflon felt. The central portion of the polyvinyl sponge is excised (Fig. 4) leaving a rim of compressible sponge lying against the septum. Flow

*Available from Mr. Norman C. Jeckel, United States Catheter and Instrument Corp., Glens Falls, N. Y.

temporarily continues through the central portion of the patch until the mesh becomes closed over with fibrin and, later, endothelial tissue. Thus, a gradual closure of the defect is effected. This is similar to Lillehei's perforated-compressed-polyvinyl-sponge technique. It is simpler than creating a temporary artificial ductus.[5]

2. *Smaller defects around 1 cm. in diameter with strong fibrous rims:* For these cases, the Mersilene sutures are placed in the tissue in the same way as for the large defects. However, the needles are passed tangentially through each side of a cigarette-shaped, noncompressed polyvinyl sponge ⅜ of an inch in diameter (see Fig. 2). The sutures are placed near the edges of the sponge so that, when they are tied down, some of the sponge is wedged between the edges of the defect. Thus, good closure is achieved without producing excessive tension on the septal tissue (see Fig. 2). The excess polyvinyl sponge is cut away, leaving a small, compact tampon in and over the defect. This method is similar to that reported by Kirklin.[1]

DISCUSSION

Age Range.—The average age of the patients in this series was slightly under 6 years, with extremes of 3 months and 33 years. Cardiac by-pass was deliberately avoided in patients under 1 year of age because of the high mortality in this age group. Instead, a two-stage approach has been employed for those infants requiring surgery to prevent recurrent heart failure and repeated respiratory infections. The first stage consists of banding the main pulmonary artery with a Dacron cloth band* to reduce the left-to-right shunt. When the child is older and can well tolerate open-heart surgery, the constriction in the pulmonary artery and the septal defect are corrected.[6]

Size and Location of Defect.—The largest defects were approximately 25 mm. in greatest diameter. Twenty-eight were located in the posterior septum behind the septal leaflet of the tricuspid valve. Eighteen were located in the outflow tract below the medial limb of the crista supraventricularis. Three were found immediately beneath the pulmonary valve. In only four of those located in the posterior septum was it necessary to transect (and resuture) a papillary muscle in order to gain good access to the area. This was done early in our series, but with increased experience and the adoption of small atraumatic needles, it is no longer used. In 4 patients, there were multiple ventricular septal defects: 3 had two, and 1 had eight, separate defects.

Associated Defects.—Three patients in our series had a patent ductus arteriosus. Two of these were closed at the same time. In one child, with equal pulmonary and aortic pressures, the ductus was temporarily occluded during the cardiopulmonary bypass while the ventricular septal defect was being closed, but afterward was allowed to remain open. This was done as part of a two-stage approach to the problem of severe pulmonary hypertension. The ductus was successfully closed at an operation undertaken 6 months later.[5]

*Seamless tubing ("Helanca Dacron warp"), 8 mm. width. Available from John Sidebotham, Inc., 4317 Griscom Street, Frankford, Philadelphia 24, Pa.

Four patients had atrial septal defects of the secundum type, varying from about 5 mm. to 15 mm. in diameter. The presence of this defect was suspected by the appearance of bright red bronchial blood passing up through the tricuspid valve after the ventricular defect had been closed. The secundum area was then visualized through the tricuspid valve. The defect was then repaired with a continuous double row of 3-0 atraumatic Mersilene suture through a suitable atrial incision.

In two instances, a pulmonic valvular stenosis was also present. Cardiac catheterization demonstrated a pressure drop across the valve, but the stenosis was of such a moderate degree that a large left-to-right shunt persisted. These cases were classified as interventricular septal defects with pulmonic stenosis rather than as acyanotic examples of tetralogy of Fallot. No cases of an infundibular stenosis are included in this series.

Two patients were discovered to have aortic insufficiency following closure of their interventricular septal defects. In 1 case, the septal defect was very large (25 mm.) and was associated with a large left-to-right shunt. Because the aorta was very small, one wonders whether there was actually a leaflet defect present or whether the appearance of valvular incompetency was due to dilatation of the aortic ring from the increased systemic flow. The other patient's defect was located above the medial limb of the crista supraventricularis just below the pulmonic valve. Her insufficiency is the most severe of the two. This, together with the location of the septal defect, suggests that there is probably an associated defect in one of the aortic leaflets.

There was 1 patient who had congenital mitral insufficiency and 3 with tricuspid insufficiency. The child with mitral insufficiency had pronounced clubbing and deficiency of the posterior leaflet of the mitral valve. She also had severe vascular changes in the small muscular arteries. The 3 patients with tricuspid insufficiency had, in effect, a left-ventriculo-right-atrial shunt. This resulted from a cleavage of the septal leaflet of the tricuspid valve. In each instance, the valve leaflet was repaired along with the septal defect.

Persistent Shunts.—There are 2 patients in whom sizable left-to-right shunts persist. These should be regarded as operative failures and will require re-operation. One of these was a 1 cm. defect closed only with interrupted atraumatic silk sutures. In the other, a large defect, a double-thickness patch of Teflon cloth was sewn in place employing a mattress suture technique as described earlier. It is believed that in the first patient, the suture line disrupted (at least partially) after cardiac contraction had resumed. The second failure may have been due to the inability of the Teflon cloth to apply itself snugly to the septum. Both of these patients were early cases who underwent operation before the present technique was evolved.

In 3 patients, there is a residual Grade 1-2 systolic murmur over the pulmonic area. They may be due to turbulence in the outflow tract (from dilatation of the pulmonary artery), from a residual leak in the repair, or possibly, from an overlooked second very small defect. In any event, the salutary clinical

response and the return toward normal of the electrocardiogram, roentgenogram, and hilar activity suggest that these murmurs are physiologically inconsequential.

Mortality.—There were 9 deaths in this series. Seven had high pulmonary vascular resistance as evidenced by catheterization studies showing essentially systemic pressures in the pulmonary circulation and predominant right ventricular hypertrophy on the electrocardiogram. All but one occurred during the first 6 months of our open-heart program. Initially, these deaths were blamed on the vascular changes because they all seemed to die a respiratory death. Pulmonary edema and interalveolar hemorrhage were prominent at autopsy. In retrospect, many of the deaths probably should be attributed to the techniques in use at that time. The addition of serum albumin* to the perfusate in sufficient quantities to balance osmotically the saline used in the donor bottles and in priming the pump has contributed importantly to the elimination of these pulmonary complications.[7] Tracheotomy is now rarely needed.

Two deaths occurred in the moderate hypertensive group. In both, the hypertension was due to large pulmonary flow. One was 18 months old and the other 3 months old. The former had a large ductus and an atrial septal defect. Pulmonary edema was found at autopsy. This is a preventable death. The 3-month-old infant now would not be submitted to cardiopulmonary bypass, but managed in two stages as described.

There was one death in the nonhypertensive group. This child, 24 months old, died unexpectedly 24 hours postoperatively following a tachycardia of 180. Presumably, this passed into ventricular fibrillation. No explanation could be found at autopsy.

CONCLUSIONS

Heart block complicating the surgical repair of interventricular septal defect is due to direct injury to the septal area bearing the conduction pathways. This injury may be produced during or following the improper placement of sutures or, perhaps, by pressure on the septum by a hard patch material. The presence of high pulmonary vascular resistance has not influenced the incidence of heart block or heart arrythmia.

In our series, the mortality is primarily related to the psysiologic consequences of a high resistance in the pulmonary vascular bed. In those cases in which the pulmonary hypertension is due to increased flow, no special measures for correction of the defect are required. However, in patients with advanced changes in the small muscular pulmonary arteries and minimal left-to-right shunt, a gradual closure is helpful in improving immediate postoperative survival. The ultimate course of these patients will depend upon the degree of restitutions that will take place in the media and, particularly, in the intima. Following repair, the situation becomes similar to the pathologic physiology of

*Kindly supplied by Dr. Paul W. Yost, The American Red Cross Blood Program.

the patient with severe pulmonary vascular changes secondary to long-standing mitral stenosis. Persistence of a high pressure in the pulmonary arterial bed may further aggravate the changes in these vessels so that regression cannot occur.

SUMMARY

A surgical technique is presented which has successfully avoided the complication of heart block in all cases of interventricular septal defect. This injury can be produced by the improper placement of sutures or, we believe, by pressure from a hard patch material. Therefore, it is important to avoid incorporating the conduction pathways in the sutures and to prevent crushing the conduction area against hard patch material. This method of placing the sutures avoids encircling the fibers, and the two-component patch eliminates the need for excessive pressure by the patch against the septum. The patch can be modified to effect a gradual closure in patients with high pulmonary vascular resistance.

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CONGENITAL DIAPHRAGMATIC HERNIA IN THE ADULT

A New Technique in the Closure of the Bochdalek Hernia

Elliott Michelson, M.D., Baltimore, Md.

HERNIATION through the space of Bochdalek is the most common type of congenital diaphragmatic hernia. This defect is usually seen in infancy but, while occurring much less frequently in the adult, it remains a clinically significant group. Bowditch,[1] in 1853, analyzed 88 patients with diaphragmatic hernias and found that 9 of the 22 with the congenital type lived after the second year of life without surgical intervention. This would suggest that a small number of these patients surviving the high mortality during the first year of life may live to a significant older age. Liebow[2] cites 9 cases of massive congenital diaphragmatic hernias in which the patients lived to the ages of 17 to 50 and suggests that long life is not incompatible with this condition. This report is concerned with the recognition and treatment of the Bochdalek hernia in the adult.

The diaphragm is a complex organ made up of two major components embryologically, which shift their position during fetal life under different time schedules of development. The upper or ventral portion of the diaphragm is formed by the septum transversum which develops in the cervical region of the paired third and fourth myotomes and migrates caudad to the first lumbar segment in the 2-month embryo. The transverse septum then fuses with the ventral mesentery. The dorsal portion of the diaphragm develops from the paired pleuroperitoneal membrane which is derived from the pulmonary ridge. Medially, the membrane fuses with the dorsal mesentery to form the posterior and central portion of the diaphragm and contains the esophageal hiatus. Mesodermal cells derived from the receding wolffian bodies fuse to form the right and left crura of the diaphragm. The pleuroperitoneal canal is closed by concentric growth of the membrane at the end of the seventh week of fetal life and complete development of the diaphragm is accomplished by fusion of this membrane with the septum transversum and derivatives from the lateral chest wall (Fig. 1). Arey[3] believes that the closure of the pleuroperitoneal communication is not due to active growth of the membrane but rather by passive expansion caused by growth of adjacent lungs, liver, and adrenals. The Bochdalek hernia results from incomplete closure of the pleuroperitoneal canal. In

From the Departments of Surgery, Sinai Hospital of Baltimore, Inc., and the Johns Hopkins University School of Medicine, Baltimore, Md.

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the tenth week of fetal life, viscera return from the umbilical cord to the abdominal cavity and, if the canal is open, loops of bowel will herniate into the pleural cavity. The small bowel having greater mobility and lesser bulk, enters the pleural space first, followed by the splenic colon. Stomach is the last to herniate, rotating 180 degrees so that its fundus lies cephalad. The Bochdalek hernia contains no peritoneal sac and is therefore a false hernia. A true hernia developing as a result of weakness of the fused pleuroperitoneal membrane is a rare condition.

Paul and Kanagasuntheram[4] find a definite sequence of bowel herniation in all Bochdalek hernias. From an analysis of two autopsies and a study of all other existing reports they find that the hernia is due to incomplete closure of the pleuroperitoneal canal with orderly sequential herniation of small bowel, followed by splenic colon and stomach. They observe that no Bochdalek hernia

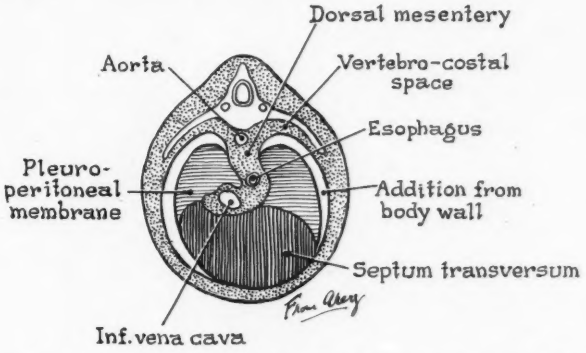

Fig. 1.—Anlagen of the diaphragm. Note that Bochdalek space is contained between twelfth rib and septum transversum.

has ever been reported with only stomach present in the left chest. The Bochdalek space is between the twelfth rib and the septum transversum and not between the twelfth rib and the vertebrae as Keith states[5] (Fig. 1). When the left diaphragmatic crura, as well as the pleuroperitoneal membrane, is underdeveloped, the hiatus will extend from the vertebrae to the septum transversum. Herniation of the kidney and adrenal may then be present. It is apparent that repair of this defect will be somewhat different from that of the typical Bochdalek hernia.

The pleuroperitoneal hernia may be small and localized close to the ventral septum or encompass the entire posterolateral portion of the diaphragm. The most common anomalies associated with this type of hernia are incomplete rotation of bowel and incomplete development of the left lung.

CASE REPORT

B. H., a 56-year-old white woman, was first seen on Oct. 31, 1955, complaining of marked dyspnea, fatigue and pains in the left chest, back, and neck. For a number of years, she had had epigastric and left upper quadrant pain associated with marked eructation, nausea,

and vomiting. Nine years before, an abdominal laparotomy had been performed at another hospital. Repair of a left diaphragmatic hernia was said to have been attempted through a 6-inch incision over the left tenth rib, but no operative report was recorded. Since this operation, she has noted increased dyspnea on exertion, but her digestive symptoms have remained unimproved. In 1952, x-ray studies of the stomach revealed displacement of the stomach and small and large bowel into the left hemithorax. The patient has always been somewhat fatigued and short of breath.

On physical examination she did not appear to be acutely ill and exhibited no respiratory difficulties at rest. The right lung was clear to percussion and auscultation, and the right diaphragm moved well. The percussion note over the left hemithorax was tympanitic,

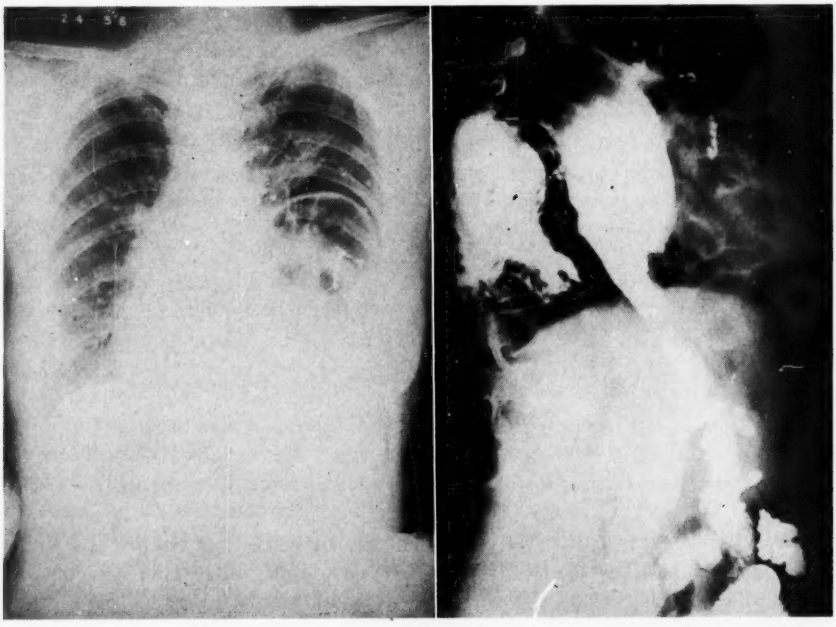

Fig. 2. Fig. 3.

Fig. 2.—Chest film following pneumoperitoneum showing left pneumothorax, dilated stomach, barium streaks in large bowel, scalloped border of colon, compressed left lung, and mediastinal shift.

Fig. 3.—Gastrointestinal series reveals stomach rotated 180 degrees within left pleural space with small bowel anteriorly placed and colon filled with air at apex.

no breath sounds but active peristaltic sounds were heard. No retrocardiac dullness could be delineated. There was a well-healed 6-inch scar over the left tenth rib. The abdomen was soft with no masses, but increased peristaltic activity was noted.

Roentgenographic examination of the chest revealed the heart and mediastinum to be shifted toward the right due to the presence of colon, small bowel, and stomach in the left hemithorax (Fig. 2). There were multiple linear strands of atelectasis at the left apex. The right lung appeared normal. Barium studies revealed rotation of the stomach through 180 degrees with the fundus placed cephalad at the level of the third rib (Fig. 3). The splenic flexure was herniated into the left hemithorax, rising to the apex of the chest (Fig. 4). Following pneumoperitoneum, air was noted at the apex of the left chest with both colon and stomach displaced downward.

The patient was admitted to Sinai Hospital on Jan. 22, 1956, for repair of a left Bochdalek diaphragmatic hernia. Through a left thoracotomy approach, a large posterolateral defect of the left diaphragm was found (Fig. 5). The anterior or ventral portion of the diaphragm was thick and contracted. A small, thick ridge of tissue was present along the twelfth rib. The esophageal hiatus was normal and the left crura was evident. There were no adhesions binding the stomach, or small and large bowel to the pleural wall and reduction was accomplished without difficulty. The apical, posterior and anterior segments of the left upper lobe appeared normal, contained carbon pigment, and expanded freely, but the entire left lower lobe and the lingula of the left upper lobe were flesh pink, without pigmentation and large blebs were seen along the periphery of these segments. Repair of the outer half of the diaphragmatic defect was accomplished by approximating the thick ventral leaf to the short muscular ridge and periosteum overlying the twelfth rib. A large

Fig. 4.—Barium enema study showing malplacement of colon in left chest.

defect, measuring 6 cm. in diameter, was still present following this partial repair (Fig. 5, *A*). Sutures placed through the muscular edges close to the esophageal hiatus tore out with each strong pulsatile thrust of the heart. In an attempt to free the diaphragm from this abnormal motion, the anterior, left lateral, and posterior attachments of the pericardium to the diaphragm were freed, and it was then possible to suture together the remaining edges of the defect (Fig. 5, *C*). Numerous blebs in the lower lobe were clamped and their base sutured. The left lung was easily inflated and filled the entire left hemithorax. The postoperative course of the patient was marked by repeated spontaneous pneumothoraces, apparently due to recurrent bleb ruptures, and this complication necessitated several aspirations and tube drainage. On discharge, 2 weeks after the operation, she felt well and had no digestive symptoms or dyspnea. On her last visit in January, 1959, she felt well, had no digestive symptoms, and stated that for the first time in her life she was able to shop without fatigue or dyspnea. Her left lung was well expanded (Figs. 6 and 7) and there was definite, though slight, impairment of motion of the left diaphragm.

DISCUSSION

Symptoms.—Cardiorespiratory and digestive symptoms associated with the Bochdalek hernia are due to the compression and distortion of the herniated bowel as well as lung and mediastinal structures. Volvulus with obstruction and perforation may occur within the hemithorax and present abdominal symptoms but without localizing signs of peritoneal irritation. In the present case, the patient had ventilated her right lung together with a small portion of the

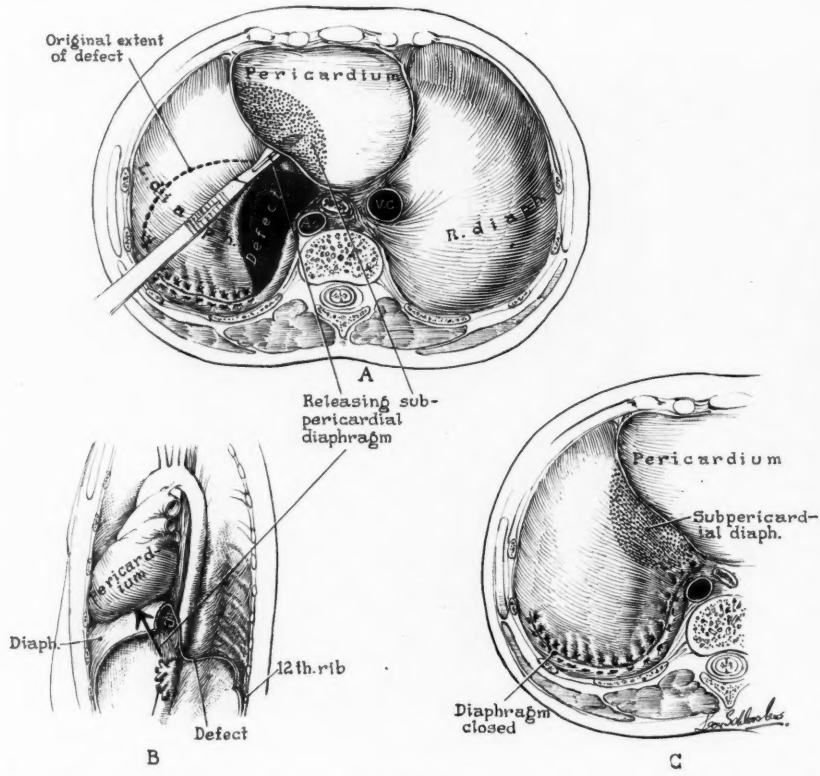

Fig. 5.—*A*, Appearance of large Bochdalek hernia after reduction of bowel. Note posterior muscular ridge and pericardial attachments. *B* and *C* show method of repair utilizing posterior muscular ridge and ventral portion of diaphragm after pericardial attachments are released.

left upper lobe. The bleb formation, absence of pigmentation, and recurrent pneumothorax suggest maldevelopment and congenital atelectasis of the lingula and left lower lobe. Recurrent incomplete volvulus of the bowel gave rise to the abdominal pain, nausea, and vomiting.

Diagnosis.—Inspection of the chest will reveal decreased expansion of the affected side. The percussion note may be either dull or tympanitic depending upon whether the herniated bowel contains predominantly air or fluid. The heart is displaced to the right and no retrocardiac dullness is noted. When

intestinal sounds are heard within the chest, the correct diagnosis is evident. In the presence of a typical history of intestinal obstruction, a flat, nontender abdomen should suggest the possibility of a diaphragmatic hernia.

Roentgenographic examination of the chest shows displacement of the heart and mediastinum to the right. Fluid levels will usually be seen which might be incorrectly interpreted as encapsulated hydropneumothorax. Lateral x-ray films and barium studies will usually reveal the stomach to be lateral to the pericardium and the splenic flexure at the apex of the chest. Pneumoperitoneum will reveal the absence of a hernial sac.

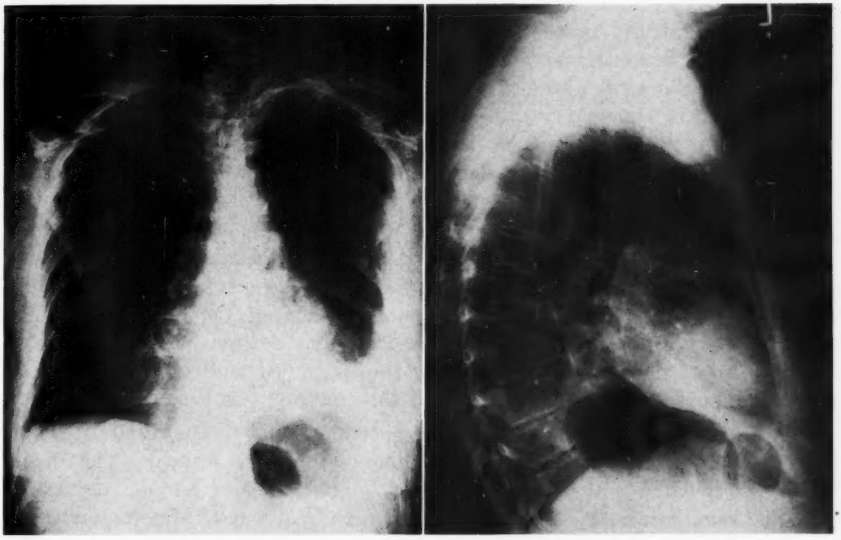

Fig. 6. Fig. 7.

Figs. 6 and 7.—Chest films made 2½ years after operation showing well-expanded left lung with bowel below diaphragm.

Treatment.—Repair of the Bochdalek hernia in the adult is usually an elective procedure although the presence of such complications as intestinal obstruction or perforation makes immediate correction necessary. In all cases, the transthoracic approach should be used. The bowel should be decompressed prior to operation. In the absence of previous infection, no adhesions are noted binding the bowel to the chest wall and reduction of the bowel into the abdomen is a simple procedure.

Numerous methods have been used in the repair of the Bochdalek hernia.[9, 10, 11] Thoracoplasty has been utilized in order to decrease this space and allow dorsoventral approximation, but ventilation of the lung is thereby lost. The latissimus dorsi muscle has been used[7] to close the lateral defect of the diaphragm and this is probably of little value since muscle atrophy will take place as the muscle moves across the exposed rib. Rives and Baker[8] have

suggested the approximation of the transversalis fascia to the dorsal edge of the diaphragm, but it is difficult to see how sufficient fascia would be available to accomplish this. Free fascia or fascia lata has been used but may be expected to exhibit paradoxical motion, may eventrate, and even atrophy. Adler and Firmie[12] have studied plastic materials and found nylon net or mesh to be tough, durable, inert, and somewhat pliable. This material can be used only as the partitioning membrane and obviously will not take part in the diaphragmatic motion. Tantulum mesh has been used but is known to fragment in time. Steel mesh may not fragment but is too rigid and may bring injury to contiguous bowel or blood vessels.

In small and medium defects of the pleuroperitoneal membrane, closure may be accomplished by the use of coapting sutures of nonabsorbable suture material. If there is marked diaphragmatic activity, injection of procaine (1 per cent) about the phrenic nerve has been suggested to produce temporary paralysis, but is unnecessary when the anesthesiologist can control its movement. In the large circular defects having a complete circle of muscular tissue, the repair of the lateral edge may be accomplished by sutures opposing the ventral muscular ridge with the tissue about the twelfth rib. The paraesophageal area, however, may be impossible to close and it is this type of defect in which repair can be accomplished by freeing the pericardial attachments to the diaphragm. Release of the anterior, left lateral, and posterior attachment of the pericardium allows the ventral or septum transversum portion of the diaphragm to be brought down and sutured to the posterior costal portion of the diaphragm. The release of the pericardium removes the continued motion of the heart on the suture line (Fig. 5). The case cited accomplished this type of repair.

When there is absence of the pleuroperitoneal membrane, as well as loss of a vertebrocostal margin, no posterior muscular ridge is present. This defect may be associated with the presence of a high kidney or adrenal herniating into the hemithorax. Prosthetic materials such as steel mesh, Dacron, or Orlon sheeting have been suggested but are either too rigid or exhibit paradoxical motion with respiration. Repair of this defect may be accomplished by elevating the remaining ventral portion of the diaphragm and suturing it to the preformed intercosto-periosteal muscle flap which may be developed from the posterolateral aspect of the fifth and sixth ribs. We have experimentally produced this defect and accomplished this type of repair in dogs,[6] with subsequent return of normal diaphragmatic motion.

SUMMARY

Congenital diaphragmatic hernias of the Bochdalek type are rare in adults but are found in a clinically significant group of patients. Diagnosis usually depends upon symptoms arising from distortion of bowel, compression of the left lung, displacement of the heart and mediastinum, and x-ray evidence of bowel herniation. Repair of the large hernias have not been gratifying in the past due to the lack of approximating muscular tissue. In very large posterolateral Bochdalek hernias, without a rim of posterior muscular tissue, elevation

of the diaphragm with suture of the ventral septal tissue to a periosteo-intercostal muscle flap at a higher rib level is suggested. When large medial defects are present, the removal of pericardial attachment to the diaphragm will allow secure closure. A successful case of this type is reported.

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CONGENITAL ESOPHAGORESPIRATORY FISTULA IN THE ADULT

Frank T. Lansden, M.D., and William H. Falor, M.D.,
Akron, Ohio

UNTIL recent years, communications of the tracheobronchial tree and the esophagus were associated with a high degree of morbidity and mortality. The advent of antibiotics and improved surgical, anesthetic, and radiologic facilities has lessened this risk and resulted in the recovery of many of these unfortunate patients.

Congenital fistulas between the esophagus and the tracheobronchial tree have been recognized for many centuries. The earliest report of such a malady is said to have been made by Gibson as early as 1696. Many reports of varying types of congenital anomalies of the esophagus followed. Most were associated with a high, if not 100 per cent, mortality.

In 1929, Vogt[1] suggested a classification of esophageal atresia which gained wide acceptance and did much to clarify the varied forms of this anomaly. Three main types were described:

I. Agenesis of the esophagus.
II. Atresia of the esophagus with no communication with the trachea.
III. Atresia of the esophagus with fistula communicating with the trachea or (rarely) with one of the main bronchi.
   A. Fistula between the proximal esophageal segment and the trachea. The cephalad end of the distal esophageal segment is closed and extends caudad from about the level of the bifurcation of the trachea.
   B. The proximal esophageal segment ends in a blind pouch. The distal segment of the esophagus communicates with the trachea or (rarely) with one of the main bronchi.
   C. Both proximal and distal esophageal segments communicate with the trachea.

The surgical techniques for the correction of these anomalies developed rapidly after reports of the lifesaving procedures developed simultaneously by Ladd[2] in Boston and Leven[3] in St. Paul. The first successful primary anastomosis of the esophagus for esophageal atresia by Haight[4] then pioneered the present surgical treatment.

From the Akron City Hospital, Akron, Ohio.
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In 1946, Holt[5] reviewed the problems of esophageal atresia and tracheoesophageal fistulas and suggested that a fourth type be added to Vogt's classification, namely: esophagorespiratory fistula without atresia of the esophagus. While this is an uncommon malady in infants, it is rarer still in adults, with only 13 documented cases reported in the literature to date.[6-13]

The major clinical and physical findings have been related to the inevitable pulmonary sepsis which develops with this lesion. Most patients have histories of repeated bouts of pneumonia and cough, often producing purulent sputum.

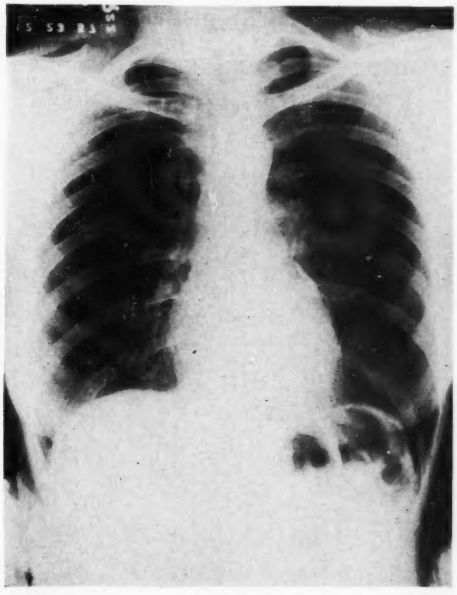


Fig. 1.—Chest roentgenogram (Case 1) which shows chronic peribronchial infiltration but no evidence of consolidation or pneumonitis. The patient had had repeated bouts of pneumonia and dyspnea.

Food particles may be raised with the sputum and hemoptysis is a common complaint. Whether or not this is secondary to the commonly found bronchiectasis, or merely a manifestation of the severe chronic bronchitis, is open to speculation. The relative rarity of this lesion has prompted the present report of congenital esophagorespiratory fistulas in 2 adult patients in whom the diagnosis was proved by surgery or postmortem examination.

CASE REPORTS

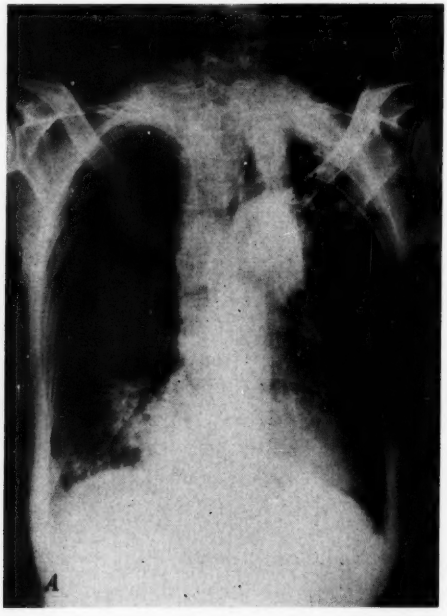
CASE 1.—M. W., a 63-year-old white woman, was admitted to the thoracic-surgical service of Akron City Hospital on Dec. 22, 1957, with chief complaints of chronic malaise, cough, and recurrent pneumonia (Fig. 1). The patient stated that she had been short of breath as long as she could remember and that she had been a "blue baby" at birth. She had frequent bouts of wheezing and cyanosis as a child and was treated on innumerable occasions for pneumonia. "Choking spells" were related to the ingestion of food and

liquids but there had been no hemoptysis and no food particles were ever raised. Copious quantities of whitish sputum were raised with postural drainage. In 1941, following a complete examination including bronchoscopy and esophagoscopy, a diagnosis of high-lying tracheoesophageal fistula was made. Surgical treatment was considered but turned down because of the apparent high risk.

The past medical history was notable only in that it included diphtheria at an early age, and hysterectomy with appendectomy at age 33. The family history was noncontributory.

Positive physical findings were limited to the chest where fine crepitant râles were heard posteriorly. There was considerable wheezing with respiration but no dyspnea was present. The vital signs were normal.

Laboratory studies revealed a chronic urinary tract infection. The fasting blood sugar, blood urea nitrogen, and hematologic studies were within normal limits.



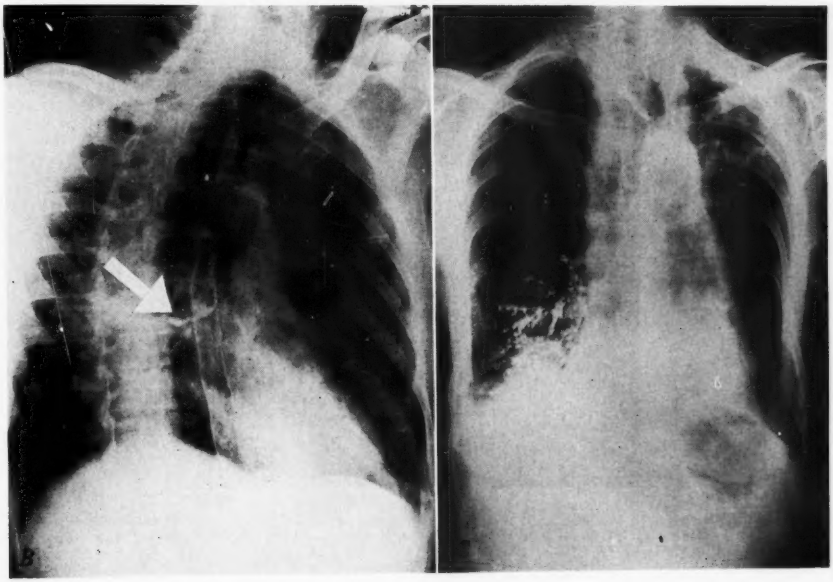
A.

Fig. 2.—*A*, Roentgenogram of the chest (Case 2) which shows chronic and acute pneumonic process involving the right middle and lower lobes.

At esophagoscopy and bronchoscopy on Dec. 24, 1957, under local anesthesia, no fistulous tract could be identified. On December 27, the patient was given a swallow of iodized oil but again the fistula could not be demonstrated. Only a moderate irregularity of the esophageal mucosa was noted at the sternal notch. On Dec. 30, 1957, bronchoscopy and esophagoscopy were repeated and a small, high-lying, tracheoesophageal fistula was found at the level of the suprasternal notch. A curved director was passed through the fistula and was visualized in the esophagus. On Jan. 6, 1958, under endotracheal anesthesia, the patient was operated upon by one of us (W. F.) for closure of the defect. A low collar incision was made through the skin and platysma muscle and the strap muscles were separated. The right sternocleidomastoid muscle was retracted laterally and the right inferior thyroid artery was divided at its origin. The right lobe of the thyroid gland was mobilized and retracted medially along with the right recurrent laryngeal nerve. The trachea was freed by blunt dissection and a small tracheoesophageal fistula was exposed. It lay at the

level of the fourth cartilaginous ring and was quite short, measuring 1 cm. in length by perhaps 7 mm. in transverse diameter. Since the lesion was extremely short, it was decided to ligate the fistula in continuity. By utilizing the sterile bronchoscope, a soft esophageal dilator was threaded through the fistula as a guide and two 2-0 silk suture ligatures were placed around the fistula. These were tied down after removal of the guide. The wound was closed anatomically and it healed without incident. The patient improved symptomatically and has remained well to date.

CASE 2.—C. J., a 60-year-old white man, was admitted to Akron City Hospital with complaints of bloody diarrhea alternating with constipation of several months' duration. He subsequently was found to have carcinoma of the rectum and underwent a combined abdominal-perineal resection. During the fluoroscopy accompanying an upper gastrointestinal x-ray study prior to surgery, a large bronchoesophageal fistula was noted which connected the esophagus with the right main-stem bronchus (Fig. 2, *A*, *B*, and *C*). Closer questioning of the patient at this time revealed a history of pulmonary sepsis for over 40 years. He stated that he had been chronically ill as a child and had repeated bouts of pneumonia



B. *C.*

Fig. 2. Cont'd.—*B*, Oblique view of the chest (Case 2) which shows passage of radiopaque medium from the esophagus into the large bronchoesophageal fistula to the right main-stem bronchus.

C, Roentgenogram (Case 2) after ingestion of radiopaque medium. The bronchogram thus produced showed massive bronchiectasis and chronic pulmonary suppuration of the right lower and middle lobes.

coupled with chronic cough and fever. There was a past history of diphtheria at the age of 8 years. He was told by a physician, at that time, that he had "ulcers on his bronchial tubes." He stated that he would "almost strangle" with pharyngeal secretions at night, and that these symptoms were worse when lying on his right side. The patient had been treated on eight occasions for pneumonia and had had three or four extensive pulmonary hemorrhages. He had been jaundiced in 1945 and had been treated for chronic gall bladder disease since that time.

Because of the x-ray findings, bronchoscopy and esophagoscopy were performed under local anesthesia. The fistula was readily seen during both examinations. The mucosa of

the tract, as well as that surrounding it, seemed grossly normal. The tract arose from the anterolateral aspect of the esophagus and communicated with the right main-stem bronchus near an accessory bronchus to the right upper lobe.

The patient was discharged and ultimately readmitted with terminal carcinomatosis. The autopsy revealed massive metastatic carcinoma in the liver and abdominal lymph nodes. The previously described fistula was seen to connect the esophagus and the right main-stem bronchus (Fig. 3). The tract was well defined and appeared to be of congenital origin. There was accompanying long-standing right lower lobar pulmonary suppuration with abscess, bronchiectasis, and pulmonary fibrosis.

Fig. 3.—Photograph of the autopsy specimen in Case 2. The large bronchoesophageal fistula is seen extending into a large area of necrotic pulmonary parenchyma.

SUMMARY

Congenital esophagorespiratory fistula in the adult is a very rare anomaly with only 13 documented cases having been published to date. The disorder is accompanied by severe long-standing pulmonary sepsis and is usually diagnosed by careful history and often as an accidental finding. Two additional cases of fistulous connection of the esophagus and tracheobronchial tree in adult patients have been presented. Esophagorespiratory fistula without esophageal atresia may be compatible with long-term survival and may be difficult to diagnose without repeated radiologic and endoscopic examinations.

We wish to express our appreciation to Mr. John Muldowney, Akron City Hospital, for the photographs used in this paper.

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PULMONARY ALVEOLAR PROTEINOSIS

Review of the Literature and Report of a Case

Leldon P. Pitt, M.D., Philadelphia, Pa.*

IN JUNE, 1958, pulmonary alveolar proteinosis was first reported in the literature[1] and was given this title by the authors—Dr. Samuel Rosen, Chief of Pulmonary Pathology, Armed Forces Institute of Pathology, Dr. Benjamin Castleman, Clinical Professor of Pathology, Harvard Medical School, and Dr. Averill Liebow, Professor of Pathology at Yale University. The first case was actually seen by Dr. Castleman in July, 1953, the specimen having been obtained by lung biopsy which proved puzzling to him; whereupon he sent the slides to Dr. Liebow who likewise recalled never having seen such lesion. Since 1955, slides for consultation have been received by the above pathologists at an increasing rate so that by June, 1958, a total of 27 cases had been compiled. Some patients were studied in their respective institutions, but the majority were studied by slides sent from various states in this country and from three foreign countries—Turin, Italy; London, England; and Canada.

This perplexing disease is characterized clinically by an insidious onset of progressive dyspnea, cough which may not be productive but often is productive of white, yellow and, at times, blood-streaked sputum, frequent chest pains, fatigability, and later by weight loss, clubbing, and cyanosis. Temperature is normal or slightly elevated. Physical examinations in the cases studied were not remarkable except for the above signs.

The ages ranged from 2 years to 56 years, but the majority were between 20 and 50 years of age. The occupations listed were as follows: lumberyard employee,[4] carpenter, electrician exposed to smoke in the production of cannon shells, butcher, animal cage cleaner, painter, machinist, toothpaste factory worker, and welder (father of the child).

CLINICAL STUDIES

Laboratory Studies.—In general, the erythrocyte counts and hematocrits were normal or slightly elevated (hemoglobin, 15 to 18 grams). The leukocyte counts varied from normal to 20,000, probably depending upon the amount of superimposed infection. Sputa smears and cultures were generally negative for common pathogens and tubercle bacilli, as were the few lung specimens that were

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*Assistant Surgeon to Pennsylvania Hospital and Benjamin Franklin Clinic, Philadelphia, Pa.

examined for fungus. Attempts to isolate virus were unsuccessful. In 2 cases there was superimposed fungus infections—one Cryptococcus and one Nocardia, both of which terminated fatally. Skin tests for tuberculosis, histoplasmosis, blastomycosis, and brucellosis gave various reactions which were of no help in diagnosis. Total serum proteins and A:G ratios were generally normal as were cholesterol, total lipids, calcium, phosphorus, potassium, chlorides, nonprotein nitrogen, and blood sugars.

X-ray Studies.—Radiographs were quite similar in all cases, being characterized by bilateral, diffuse granular, feathery or slightly nodular infiltrations radiating from the hilum and, in general, were more extensive in the lower two thirds of lung fields. There was a notable absence of hilar adenopathy and no calcification has been observed.

Pathology.—Grossly, the lungs were heavier, firmer, less spongy, and more friable than normal. There were diffuse confluent grayish areas of infiltrations from which, in some specimens, oozed milky fluid on sectioning. The weight of some lungs was remarkable—as high as 3,500 to 4,000 grams at autopsy. Microscopically, the alveoli were either partially or completely filled with eosinophilic granular, PAS positive, proteinaceous material in which there were varying numbers of foamy mononuclear cells. The interalveolar septi were remarkably well preserved and showed essentially no cellular infiltration and no proteinaceous material. It is thought that most, if not all, of this protein material is derived from the transformation of the large mononuclear cells which line alveoli, the so-called "septal cells." Mediastinal and peribronchial lymph nodes examined showed only reactive hyperplasia and some erythrophagocytosis and were not significantly enlarged grossly.

Spectroscopic examination revealed no evidence of heavy metals and examination for radioactivity revealed none.

CASE REPORT

The patient, T. K., age 50 (U.H. No. 31683), was a white male, ex-stevedore whose chief complaint was progressive dyspnea of one year's duration. This was associated with a nonproductive cough, intermittent nonspecific chest pains and, a few months before death, by marked cyanosis and clubbing of his fingers. He had been treated by his family doctor for heart disease without improvement. There was no history of hemoptysis, previous pulmonary diseases, or peripheral edema. On the day of admission he had had several drinks of alcohol; he collapsed into unconsciousness which precipitated his initial hospitalization on Aug. 31, 1958.

Past medical history revealed no cardiovascular, renal, or gastrointestinal diseases. He denied allergy, tuberculosis, or other serious illnesses.

Social history revealed that he smoked a package of cigarettes per day and drank alcoholic beverages in large amounts at times. He had worked as a stevedore until a year before admission when he had retired because of dyspnea. There was no history of exposure to toxic fumes and dust or the consumption of oily materials.

Physical examination revealed a chronically ill, well-developed but poorly nourished man who had moderate dyspnea, cyanosis, clubbing of fingers, and coarse râles bilaterally. The temperature was 101° F., respirations 32, pulse 130, and blood pressure 110/80 mm. Hg. The heart was thought to be slightly enlarged but there was no evidence of decompensation. There was no lymphadenopathy and no enlargement of the abdominal viscera.

Laboratory data included hemoglobin 14 grams, hematocrit 42 mm., leukocytes 20,000 on admission with 79 per cent segmented neutrocytes, 19 per cent lymphocytes, and 2 per cent monocytes. After antibiotics, the leukocytes numbered 7,000 with a normal differential count. Urinalysis was normal, serum bilirubin, calcium, alkaline phosphatase, potassium, and blood urea nitrogen were normal. The phenolsulfonphthalein test showed 8 per cent retention in 45 minutes (normal 0-5), serum total proteins 7.0 grams per cent, albumin 3.5, and globulin 3.5. The transaminase varied from 28 to 52 units (normal 20-40), sedimentation rate 38 mm. per hour (normal 0-10). Multiple sputum studies were negative for tubercle bacillus and fungus but did show nonhemolytic streptococcus and *Staphylococcus albus.*

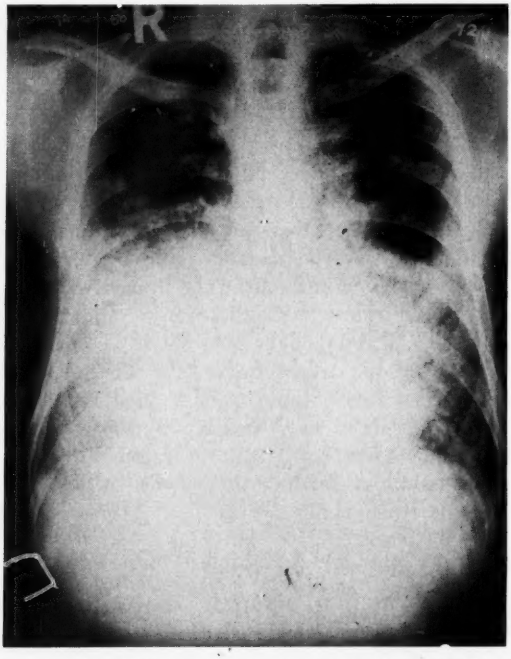

Fig. 1.—Film taken on Oct. 14, 1958, showed "soft cottony" infiltration chiefly in mid and lower lung fields.

Electrophoretic separation of serum proteins revealed hypoalbuminemia and hyperglobulinemia, the latter involving chiefly the alpha and beta fractions. The electrocardiogram showed changes consistent with subendocardial ischemia.

Skin tests for tuberculosis were positive on second strength, but were negative for histoplasmosis, blastomycosis, and coccidiomycosis.

Chest x-ray studies (Fig. 1) revealed bilateral, diffuse granular, or slightly nodular pulmonary infiltration most marked in the mid and lower lung fields and no hilar adenopathy. This was diagnosed as bronchopneumonia.

The administration of digitalis and tetracycline resulted in a return to normal temperature and symptomatic improvement, without radiographic changes in the chest. After leaving the hospital 3 weeks after admission, the patient was followed in the Out-Patient Department and was noted to have increasing dyspnea, cyanosis, weakness, and dizziness. He was readmitted on Dec. 17, 1958. A roentgenogram of the chest showed a progression of pulmonary infiltration. The following day, I performed a lung and pleura

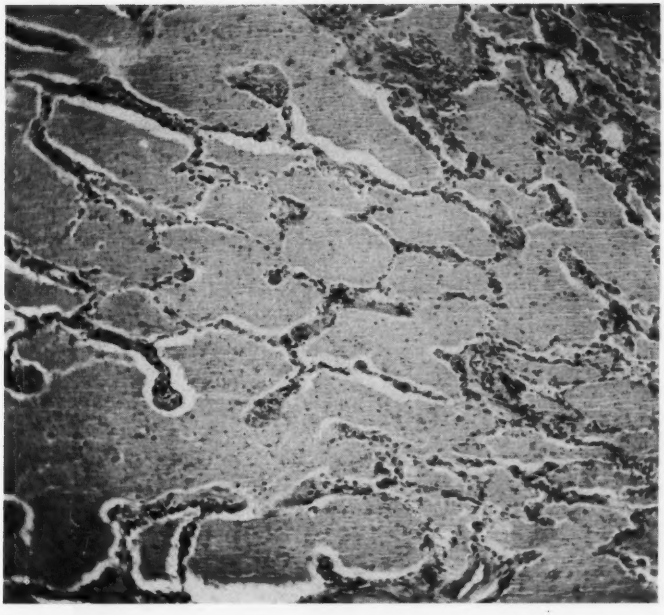

Fig. 2.—Hematoxylin and eosin preparation showing well-preserved, essentially normal alveolar septa and filling of alveoli with granular proteinaceous material. Note absence of inflammatory response.

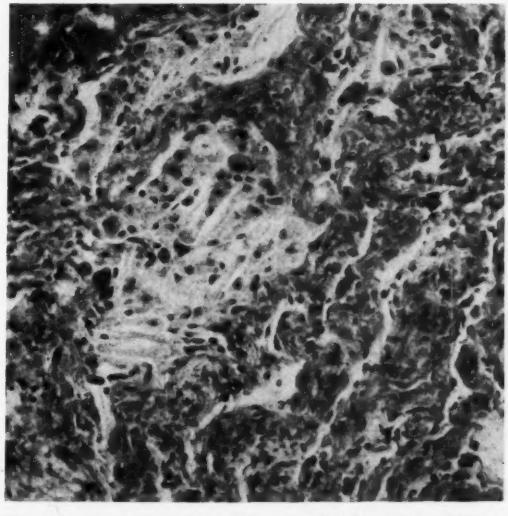

Fig. 3.—Note granular mononuclear septal cells and slit-like spaces which probably represent cholesterol crystals. Occasional multinucleated giant cells are also seen.

biopsy (Figs. 2 and 3) under 1 per cent procaine anesthesia, through the anterior right third interspace. There were fibrinous adhesions between the visceral and parietal pleurae; these surfaces were dull gray and three times normal thickness. The lung was soft and friable and, upon sectioning, a milky fluid oozed diffusely from the cut surfaces. The sutures cut through the lung parenchyma readily while closing the defect. After operation, his condition was unchanged and a roentgenogram showed no change in the chest. He again signed out 2 days after the operation. He was next seen on Jan. 8, 1959, when he was deeply cyanotic, dyspneic, and died soon after admission from respiratory insufficiency (Fig. 4).

Fig. 4.—Film taken on the day of death, Jan. 8, 1959, showed extensive densities of both lungs.

DISCUSSION

The etiology of this baffling disease remains unknown, but it is interesting to speculate in regard to the possible relationship to the inhalation of detergents, plastic fumes, and insecticides. Diseases most likely to be confused with this lesion are pulmonary edema, pneumocystis pneumonitis, and chronic interstitial pneumonia. Pulmonary edema presents a more acute picture which should respond to cardiorenal therapy. Pneumocystis infection usually occurs in premature infants who die in the second or third month of life. The roentgenogram may be quite like one of a case of proteinosis but examination of lung tissue reveals the parasites which produce this disease. Interstitial pulmonary fibrosis may be confusing clinically, but pathologically the picture is quite distinct from proteinosis. Miliary tuberculosis, sarcoidosis, and microlithiasis should offer little difficulty in differential diagnosis.

Therapy.—There is no known specific therapy as yet. Antibiotic and antifungus drugs are useful for superimposed infections but have no effect on the disease itself. Cortisone may produce temporary symptomatic improvement but has no lasting effect.

Prognosis.—Insufficient time has elapsed to gain significant data in this regard. In any case, however, prognosis should be guarded. Of the series reported, 5 patients were definitely improved at the time of report. One seemed to have fully recovered, both clinically and radiographically. In some, the symptoms improved but roentgenographic studies did not, and vice versa. Eight have died from pulmonary insufficiency. One patient died 4 months after onset, while another expired 4 years after onset of symptoms. Several patients have had negative x-ray studies of the chest from 3 months to 10 years before onset of symptoms.

Summary.—It seems unlikely that pulmonary alveolar proteinosis has been present for many years, as the pathologic characteristics are distinct and should not have been confused with previously known diseases. The onset is usually insidious and the clinical picture is characterized by progressive dyspnea, cough, often productive, and, later, cyanosis and clubbing of fingers. Radiographically, there is bilateral diffuse infiltration radiating from the hilum. Pathologically, the alveoli are filled with granular acidophilic protein material which is rich in lipids. The etiology and treatment are obscure and prognosis is uncertain. The only way to make a definite diagnosis in the living patient is by lung biopsy which can be done readily through a small intercostal incision under local anesthesia. I suggest this procedure be used more frequently for undiagnosed chronic pulmonary lesions.

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AORTIC-ESOPHAGEAL FISTULA

Walter J. Henry, M.D., and Laurence Miscall, M.D., New York, N. Y.

AORTIC-esophageal fistula occurs so infrequently that rarely does a surgeon see more than one case in a lifetime. Therefore it is not surprising that the diagnosis has been made usually at postmortem examination. This case is the only one that has been seen on two large thoracic surgical services in New York in 25 years. Although it presented the characteristics previously described in the literature, like so many others, the correct diagnosis was established after death.

CASE REPORT

O. M., a 4-year-old mentally retarded Negro girl, was admitted to the Thoracic Surgical Service of Triboro Hospital on July 21, 1958. Her chief complaints were only mild retrosternal pain which had been quite severe at its onset 3 days before admission, and dysphagia which had progressed, on admission, to the complete refusal of oral intake. A history of foreign body ingestion was denied by the patient and her parents. She appeared chronically ill with a temperature of 101° F., a pulse of 100, and a blood pressure of 120/80 mm. Hg. Her weight of 22 pounds was 15 pounds below normal. Dehydration was marked. The most prominent objective finding was substernal pain elicited by manual pressure. Examination of the heart and lungs was unremarkable. She had a hemoglobin of 14.3 grams, a hematocrit of 41.5 per cent, and a white count of 16,000. An emergency chest plate showed a foreign body, a coiled spring, in the esophagus, a right aortic arch, and minimal widening of the superior mediastinum (Fig. 1).

With a diagnosis of perforation of the esophagus and mediastinitis, thoracotomy was performed. Under general anesthesia using endotracheal technique, the right posterior mediastinum was opened extrapleurally through a paravertebral incision. Exploration revealed a right-sided aorta and the spring within the esophagus just below the arch. Its lower end had penetrated both the esophageal and aortic walls. All tissues were edematous but frank pus was not found. The esophagus was mobilized anteriorly and the sharp end withdrawn from the aortic wall and then by rotation it was removed from the esophagus. Neither bleeding nor leakage occurred after this maneuver, and therefore no attempt was made to close the minute perforations. The incision was closed with drainage of the posterior mediastinum. The foreign body was a spiral wire spring of two and one half turns with the diameter of a 20 gauge needle (Fig. 2).

The patient did well in the early postoperative period. After 3 days of parenteral feeding, oral intake was started and increased to a semisolid diet by the seventh day. The drains were removed on the seventh day. On the eighth day, after several tarry stools, the hemoglobin was 7.4 grams with a hematocrit of 26 per cent and normal bleeding and clotting times. Studies of the gastrointestinal tract revealed only some degree of malrotation. On

From the Thoracic Surgical Service, Triboro Hospital, Queens Hospital Center, Jamaica, N. Y.

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the tenth postoperative day, mild shock with hypotension and tachycardia was attributed to gastric hemorrhage after fresh and dark clotted blood was aspirated from the stomach. A suspicion of blood dyscrasia was not substantiated by further detailed study. The situation was controlled on each occasion by blood transfusions. She improved until the thirteenth postoperative day when, following several black stools, she again showed hemorrhagic shock.

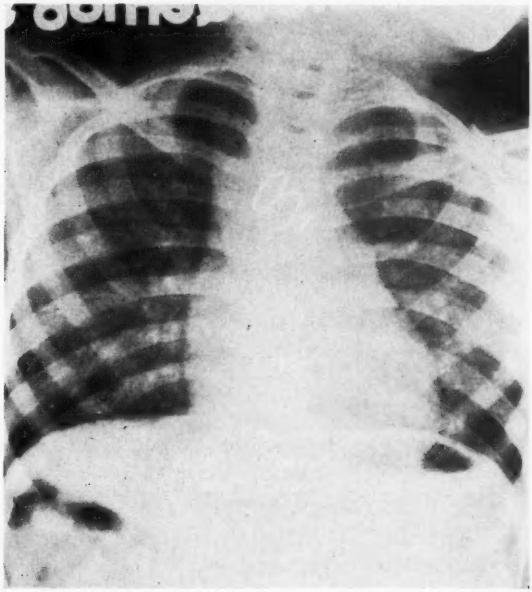

Fig. 1.—Roentgenogram showing the wire spring which perforated the esophagus and the right aortic arch.

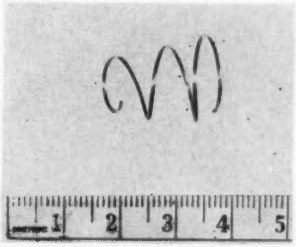

Fig. 2.—Photograph of the spiral spring.

A gastric tube was passed with particular attention to aspiration of the esophagus which did not yield blood. She again responded to transfusions. On the morning of the fourteenth postoperative day, she appeared alert and comfortable with a rapid pulse but she was not in shock. While a blood transfusion was being started she suddenly had a massive hematemesis and died.

At autopsy, a fistula between the esophagus and the arch of the right-sided aorta was demonstrated. This tract was 2 mm. in diameter and 1 cm. in length (Fig. 3). The tissue

Fig. 3.—Photograph of the opened esophagus with a probe in the fistulous tract.

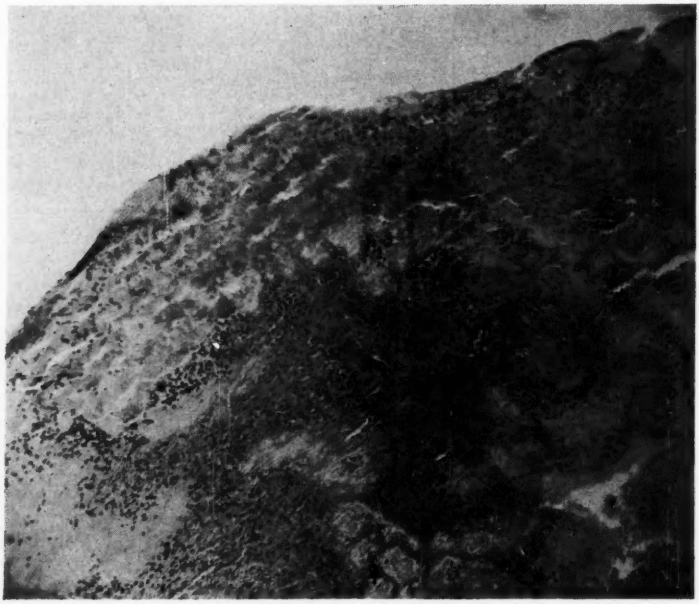

Fig. 4.—Photomicrograph of the plug of tissue removed from the esophageal orifice of the fistulous tract. Note the necrotic tissue membrane with a large number of inflammatory cells, bacterial colonies, and some squamous cells.

of the fistula was edematous and inflamed but without frank pus. Microscopic examination of this tract showed a necrotic tissue membrane with many inflammatory cells, frequent bacterial colonies, and a few squamous cells (Fig. 4). Cultures of the specimen showed a coagulase positive *Staphylococcus aureus*. Other findings were situs inversus and a stomach filled with blood.

DISCUSSION

Barrie and Townrow,[1] in a review in 1946 of the literature of aortic-esophageal fistula, found 80 reported cases. Since that time other cases have been added.[2-3] These reports indicate that the following features are quite constant. The perforation has been caused usually by a sharp object, often a fish or rabbit bone, which probably accounts for the larger number of cases reported from the British Isles where such foods are common. The aorta has been the great vessel most frequently involved due to its proximity to the area of the esophagus, where foreign bodies often lodge. After the ingestion of a foreign body, chest pain occurred in the majority of cases, receded for a variable time but generally returned before death from hemorrhage. A striking fact is the regularity with which a fatal hemorrhage has been preceded by a signal hemorrhage. Barrie and Townrow[1] in presenting 18 well-documented cases observed that all exhibited such signal hemorrhage. The interval averaged 1 day and ranged from 2 hours to 3 days. In our case, the first evidence of bleeding was 7 days prior to death. Spasm of the arterial wall closing the orifice, hypotension from hemorrhage, and occlusion of the fistula by clots, later digested with infection, have been offered as reasons for the latent period.

The sequence of events in the formation of such fistulas may follow this pattern. The esophagus is perforated by a sharp object at the aortic arch level with leakage into the mediastinum. The sharp end may either injure the aortic wall or completely traverse it, as in our case. The foreign body may pass through the alimentary tract spontaneously or be removed surgically. Sepsis follows in the wall of the esophagus and aorta and surrounding area. A fistula then evolves from infection and abetted, perhaps, by digestion from esophageal contents. The initial bleeding may exert some tamponade effect only to recur as a fatal and massive hemorrhage with further progress of infection and digestion. We have not found in the literature any case of established aortic-esophageal fistula which has been successfully treated. Even if the diagnosis were established after the signal hemorrhage, with the fistula already established, surgical treatment in the presence of such infection would be a challenging problem. In anticipation of this entity the best approach seems to be its prevention. These suggestions are offered after the experience with this patient. Foreign bodies of this type with significant symptoms indicative of possible injury to the aorta should be removed early through an open extrapleural approach if possible. This is particularly true if serious symptoms and signs of mediastinitis are present. The esophagus and aorta should be separated by the interposition of other tissues and the area drained. Even though small,

the esophageal opening should be closed by sutures and the esophageal lumen drained by tubes with constant suction. Antibiotic therapy should be broad and massive in type and should be continued for a considerable time.

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A PULSATING EPIGASTRIC MASS DUE TO PERFORATION OF A TUBERCULOUS PERICARDIAL ABSCESS THROUGH THE DIAPHRAGM

R. G. Connar, M.D., H. B. Murdaugh, Jr., M.D.,** and H. D. MacIntosh, M.D.,*** Durham, N. C.*

PERICARDIAL cysts and diverticula appear radiographically as projections from the cardiac silhouette, but a definite diagnosis is not commonly established prior to thoracotomy. They may be asymptomatic or may cause pain, fullness in the chest, and cough.[1] Extensive anatomic derangement or destruction of neighboring structures rarely occurs. The only report of such a case was published by E. H. Cushing in 1937.[2, 3] His patient had a tuberculous pericardial diverticulum which dissected through the anterior chest wall. The present report concerns a patient who also had a tuberculous pericardial diverticulum or abscess that perforated the diaphragm and presented in the epigastrium as a pulsating mass.

CASE REPORT

J. A. O., a 50-year-old textile worker, was admitted to the Durham Veterans Administration Hospital on Dec. 14, 1953, with the chief complaint of pain in his chest and abdomen. He had considered himself well until 11 weeks prior to admission when he had developed a "2-week cold" characterized by cough and left lateral pleuritic chest pain. He consulted his physician and was given symptomatic therapy. The cough subsided, but the pleuritic pain persisted and was associated with a constant dull pain along the left anterolateral costal margin which radiated into the epigastrium. After 2 weeks of continual discomfort, he again consulted his physician, and a pulsating mass was found in the epigastrium. The patient was hospitalized elsewhere. Fluoroscopic examination and x-ray studies, with the aid of induced pneumoperitoneum, revealed a pulsating 15 cm. bilobular mass in the epigastrium. The patient declined surgical exploration and returned home to bed rest. Due to persistence of pain, the patient again sought medical attention and was admitted to the Durham Veterans Administration Hospital.

The patient denied having had any chest pain prior to his present illness and had no known contact with tuberculosis. He denied having had fever, chills, or night sweats, but had lost approximately 10 pounds in weight during his present illness. There was no past history of hypertension, syphilis, intermittent claudication, or symptoms of congestive failure.

From the Departments of Surgery and Medicine, Veterans Administration Hospital and Duke University School of Medicine, Durham, N. C.

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*Assistant Professor of Surgery; Chief, Division of Thoracic Surgery, Durham VA Hospital. Present address: 1 Davis Boulevard, Tampa 6, Fla.

**VA Clinical Investigator, Associate in Medicine. Present address: Department of Medicine, University of Alabama, Medical Center, Birmingham, Ala.

***Assistant Professor of Medicine.

Physical examination revealed a temperature of 98° F., blood pressure 90/60 mm. Hg, a pulse rate of 96, and a respiratory rate of 16. Fremitus, resonance, and breath sounds were diminished at the left lung base. The heart was not enlarged laterally to percussion.

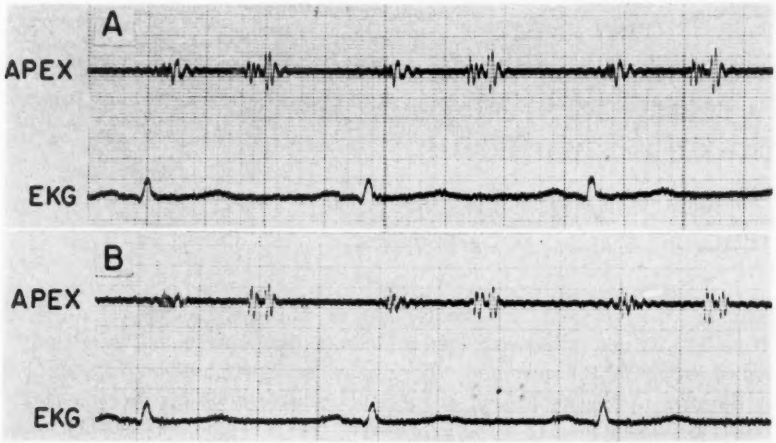

Fig. 1.—Phonocardiogram of apex. *A* is control and *B* is with application of pressure to the epigastric mass.

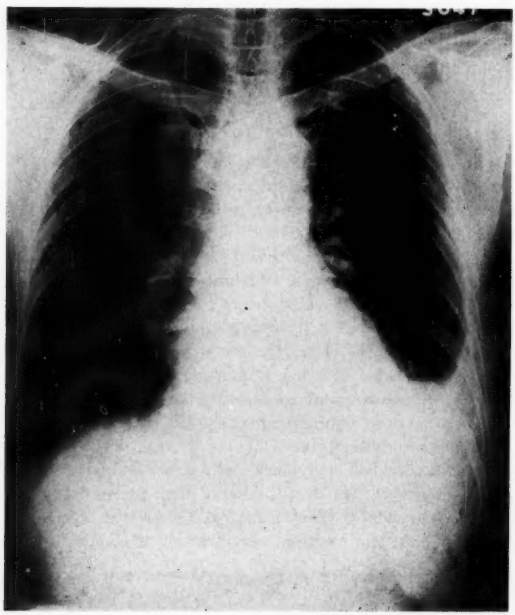

Fig. 2.—Roentgenogram of the chest on admission to the hospital.

No murmurs were heard over the precordium. The aortic and pulmonic second sounds were of equal intensity. The second sound at the apex was split. There was a 9 to 10 cm. mass in the epigastrium which pulsated paradoxically with ventricular contraction, the expansion

occurring before the carotid and femoral pulsation. The mass was dull to percussion and blended into the area of cardiac dullness. A murmur could not be heard, but there was a transmission of the second sound from the cardiac apex over the mass. The splitting of the second sound could be obliterated by exerting pressure on the mass (Fig. 1). No murmur could be heard posteriorly. The liver, spleen, and kidneys were not palpable. There was no venous distention and peripheral pulses were normal.

The Kahn test was negative. White blood cell count was 7,400 with a normal differential. Hemoglobin was 12.5 grams per cent and the sedimentation rate (Wintrobe) was 51 mm. per hour. Urinalysis was normal. A roentgenogram (Fig. 2) revealed a left pleural effusion, but a parenchymal lesion could not be seen. Thoracentesis confirmed the presence

Fig. 3.—Artist's concept of the pericardial abscess perforating diaphragm shown in midplane sagittal section. The abscess is below the heart, extends in front of the liver, displaces the stomach down, and produces the epigastric protuberance.

of a pleural effusion, the fluid being cloudy and straw-colored in appearance. The specific gravity was 1.019 and there were 4,425 white blood cells per cubic millimeter of which 90 per cent were mononuclear cells. Protein content of the fluid was 5.4 grams per cent. No tubercle bacilli could be demonstrated by either stain or culture. The cells were classified as Papanicolaou Type III. First strength tuberculin PPD skin test was positive. The electrocardiogram showed a horizontal axis and T-wave changes consistent with left ventricular ischemia. X-ray studies of the abdomen, including planigraphy and studies before and after induced pneumoperitoneum, revealed a well-outlined epigastric mass which was noncalcified and caudad to the diaphragm.

At surgical exploration on Dec. 31, 1953, the abdominal cavity was entered through an upper midline incision. A pulsating mass was encountered which was not attached to the

anterior parietal peritoneum and appeared to be covered with a layer of peritoneum. Pulsations were synchronous with ventricular contraction and quite distinct from pulsations of the abdominal aorta. The mass was adherent to the dome of the liver but could be separated readily by blunt dissection (Fig. 3). As the mass appeared to arise from above the diaphragm, the incision was extended into the left thorax through the eighth interspace. As the pleura was opened, the pleura and pericardium were seen studded with tubercles; the pleura was biopsied. The mass appeared to arise from the pericardium, displacing the anterior leaf of the diaphragm with a ventral perforation through this structure. At the point of perforation, the mass was approximately 6 cm. in diameter and no communication with the pericardial space could be demonstrated. Needle aspiration revealed thick, yellow, purulent material. The chest incision was closed, and the lower half of the abdominal incision was also closed so that the mass could be isolated from the peritoneal space. Approximately 500 c.c. of thick, yellow pus was drained, cultures of which subsequently yielded

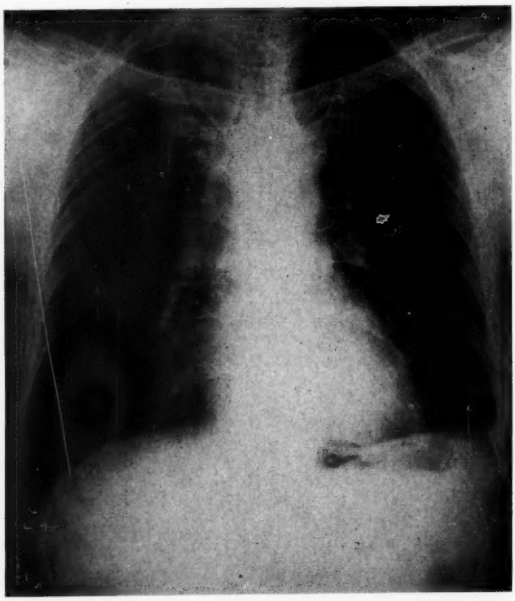

Fig. 4.—Follow-up roentgenogram of the chest.

tubercle bacilli. Drains were placed in the abscess cavity, and the upper portion of the abdominal incision was left open. The patient was started on chemotherapy receiving 300 mg. of isonicotinic acid hydrazide (INH), 12 Gm. para-aminosalicylic acid (PAS), and 2 Gm. streptomycin daily from Dec. 31, 1953, until Jan. 29, 1954, when PAS was discontinued and streptomycin reduced to 1 Gm. twice weekly. The postoperative course was uneventful. By the end of 6 weeks the abscess cavity had completely healed and the electrocardiogram returned to normal. On April 14, 1954, the patient was sent home on modified bed rest with instructions to continue chemotherapy.

Following discharge from the hospital, the patient continued to do well and remained symptom free. Streptomycin was discontinued in January, 1955, and INH in June, 1955. In September, 1955, a roentgenogram (Fig. 4) and electrocardiogram were normal, and the patient had returned to work. Our last communication was in April, 1958, at which time he was asymptomatic and working full time.

DISCUSSION

Hart[4] reported the first pericardial projection in 1837. Subsequent reports have suggested that such projections may represent a true pericardial hernia, a coelomic cyst, a congenital pericardial diverticulum, or an acquired or inflammatory pericardial diverticulum.[2, 5-9] A true pericardial hernia has been described as a protrusion of the serous membrane through a defect in the fibrous membrane of the pericardium.[10] Coelomic cysts are thought to represent a persistence of the ventral parietal recess of the pericardial coelom.[11] They are usually located at the right cardiophrenic angle and do not communicate with the pericardial space. While some authors consider pericardial diverticula as congenital in origin, others believe that they result from inflammatory processes.[2, 6, 8, 12, 13]

Tubercle bacilli have rarely been demonstrated in pericardial diverticula. Freedman[14] reported 2 such cases in 1937, but one of these had been previously reported by Cushing. Eschbach[12] in his review erroneously referred to these as 3 cases.

Tuberculosis, even with its protean manifestations does not often involve the muscle component of the diaphragm. Tu,[15] in 1950, reported what was stated to be the second case of a tuberculoma of the diaphragm. Lucke[16] describes caseous foci in the diaphragmatic muscle in military tuberculosis, and Tobias[17] reported immobilization of diaphragm with tuberculous involvement of the muscle of the diaphragm. Auerbach[18] reviewed 209 cases of tuberculosis of serous cavities and described 11 instances of spread of tuberculosis through the diaphragm to the peritoneum via the lymphatics, and 5 cases of spread from the peritoneum to the pleura by caseous foci through the wall of the diaphragm. He did not record the occurrence of perforation of the diaphragm by a supradiaphragmatic tuberculous abscess. Tuberculous abscesses of the peritoneal cavity, however, have been known to perforate the diaphragm.[19]

SUMMARY

An inflammatory pericardial diverticulum or abscess of tuberculous origin that perforated the diaphragm and presented in the epigastrium as a pulsating mass is reported. This report is of interest not only because of its rarity, but because it again demonstrates the destruction possible from an inflammatory pericardial abscess and describes an unusual type of epigastric mass.

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A COMPLICATION OF THE HUFNAGLE VALVE

A Case Report

L. Walter Fix, M.D., and Robert Riker, Captain, USAF (MC),***
Martinsburg, W. Va.

NUMEROUS attempts to remedy the defects in the cardiac-valvular system produced by the many congenital, infectious, and degenerative processes of man have been made.[1, 2, 3] It is apparent that some degree of success has been achieved[4, 5] but inadequacy of the present techniques is evidenced by the constant new approaches to the problem.[6]

A recent case studied clinically and anatomically at this hospital demonstrated a postoperative complication that does not seem to have been previously reported.

CASE REPORT

A 28-year-old white man had rheumatic heart disease with aortic insufficiency of such degree that surgical valve insert in the aorta was considered advisable. In May, 1956, the patient had a Hufnagle valve, plastic type, inserted in the descending thoracic aorta at another hospital. Recovery was excellent and the patient had one year of fairly normal life.

In May, 1957, he was admitted to this hospital with signs and symptoms of a left femoral artery embolus. An embolectomy of the left femoral profunda artery was performed. He was discharged on anticoagulants and did well. In June, anticoagulants were discontinued. In September, 1957, pain in the left calf was noted but palpable pulses and warm extremity were present. Arteriogram showed definite narrowing at site of previous embolectomy. He was discharged with the advice that a bypass operation would be performed if pain persisted.

In April, 1958, acute sudden pain developed, associated with numbness and coldness of the left leg. Dorsalis pedis and posterior tibialis pulses were absent on the left. Conservative management was administered. He recovered fairly satisfactorily with some residual claudication and was sent home on leave. On July 27, 1958, while on leave, sudden acute right leg pain was experienced. The right femoral pulse was good but dorsalis pedis and posterior tibialis vessels were without pulse. The right leg was colder than the cool left one. An aortogram showed occlusion of left common iliac at its bifurcation. A right femoral arteriogram showed occlusion of a segment of the right popliteal artery with fair filling below. On Aug. 7, 1958, a right popliteal artery embolectomy was performed. Anticoagulants were administered. Recovery from this operative procedure was uneventful.

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*Assistant Chief Laboratory, VA Center, Martinsburg, W. Va., and Associate in Pathology, George Washington University.

**Resident in Surgery, George Washington University Hospital. Currently attached to VA Center, Martinsburg, W. Va.

On Aug. 28, 1958, the patient was taken to surgery to have an iliacfemoral bypass graft inserted on the left leg. Immediately following Pentothal, Anectine, and easy intratracheal intubation, the patient developed ventricular fibrillation and died despite cardiac massage and electric defibrillation.

Postmortem Findings.—At autopsy the severe, deforming, chronic, aortic valvulitis was present. No verrucae were evident. The left external iliac and common femoral arteries had a large, fairly well-organized thrombus extending into superficial femoral and femoral profunda arteries. The attachment was firmer in the lower branches so that proximal propagation of the embolus in the iliac portion was evident (Fig. 1).

Fig. 1.—Embolus in iliac and femoral arteries.

The Hufnagle valve was in position with no ulcerations of the aortic wall or deformities other than the pressure defects from the valve fixation rings (Figs. 2 and 3). Within the valve, the ball was seen to be considerably altered (Figs. 4 and 5). Projecting from one side of the ball was an irregular friable soft reddish-purple mass which extended downward into a break in the laminated plastic ball. The ball was thinned at this side and on cut section the friable thrombus was seen to separate two of the layers of the ball. This extended beneath the layer of plastic for about one half of the diameter. The hollow ball had a well-preserved inner lamina but an outer table that was split at one site (thrombus area) and thinned over about one half of its diameter.

Fig. 2.

Fig. 3.

Fig. 2.—Hufnagle valve in situ.
Fig. 3.—Aorta with pressure rings of Hufnagle valve and bisected valve.

Fig. 4.

Fig. 5.

Fig. 4.—Ball of Hufnagle valve showing split on surface and large thrombus.
Fig. 5.—Ball of Hufnagle valve bisected. Note projection of thrombus from surface and marked thinning with separation of the outer lamina on cut surface.

Microscopic studies of the valve ball demonstrated an organized laminated thrombus without evidence of any bacterial growth or inflammatory reaction surrounding it. Sections through the embolus in the left femoral artery showed a well-organized portion at the origin of the profunda branch with a more recent thrombus above this site. Blood cultures were negative for bacterial growth.

CONCLUSIONS

Although artificial valves hold great promise in the surgical treatment of those who have valvular defects, one must select the material of which they are made very carefully. In addition, long-term follow-ups will be necessary to assay the hemodynamic consequences and deteriorative tendencies of such prostheses. In this case, the laminated plastic material finally broke down with resultant split in the surface. At this site, the ball developed a thrombus. Multiple emboli broke off from the thrombus with resultant peripheral arterial occlusion. The laminated hollow ball proved undesirable in this case because it split with resultant thrombus formation.

SUMMARY

A thrombus formed in a 28-year-old white man as a result of splitting of the lamina in the plastic ball of a Hufnagle valve. Multiple emboli from this artificial valve thrombus occurred over a period of 1½ years. Emboli in patients with artificial valves must be carefully considered as potentially arising from the valve.

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A SIMPLE OVERHEAD STERNUM RETRACTOR

John Storer, M.D., and Edward R. Malia, M.D., Cleveland, Ohio

RETRACTION of the sternum during the surgical attack on certain cardiac lesions has formerly required the concerted effort of one assistant. This, of course, applies only to the incisions making use of a transverse sternotomy. It is our practice in most cases requiring total body perfusion to make a bilateral submammary incision through the fourth intercostal space, and to transect the

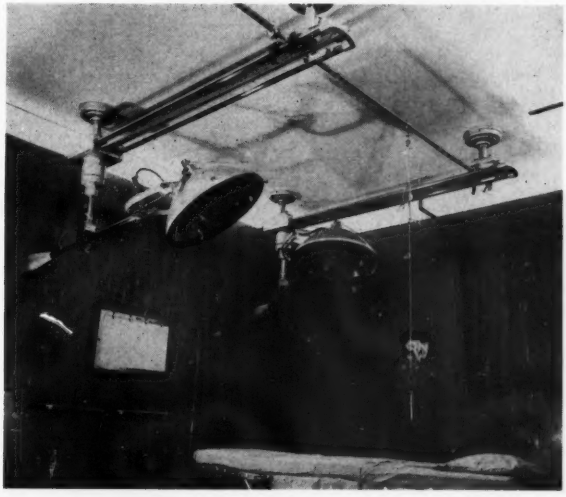

Fig. 1.—Sternum retractor assembly suspended over operating table.

sternum in V-shaped fashion. We have used a variety of retractors to spread the interspaces but all have the disadvantage of displacing the inferior portion of the thoracic cage along with the inferior portion of the transected sternum in a posterior direction. This exerts an undue amount of pressure on the myocardium and visibly affects the cardiac action. It is extremely inconvenient for an assistant to maintain continuous traction on this portion of the sternum and almost impossible over a prolonged period to keep the sternum off of the myocardium. It therefore occurred to one of us (E. R. M.) that two hooks could be inserted around the fifth rib, just lateral to the sternum, and suspended from an overhanging pulley. This implement has been used in the last 20 procedures which required this type of incision. It has proved to be completely satisfactory. The cord which is attached to the sternal hooks does not interfere with the surgical technique in any way.

From the Departments of Thoracic and Cardiovascular Surgery and Anesthesiology, Huron Road Hospital, Cleveland 12, Ohio.

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This retractor is arranged in our operating room by fixing a metal shaft to the two parallel supports for the American sterilizer dual video lights (Model D-V 22). A simple pulley and stout cord are then sterilized and just preparatory to surgery one of the completely gowned assistants fastens the pulley and cord to the overhanging rod. The operating table is situated under this rod in such a manner that the pull on the hook will be at about a 10-degree angle toward the feet. The cord is tied into a small bundle and left suspended in the air until that point in the operative procedure is reached when sternal retraction

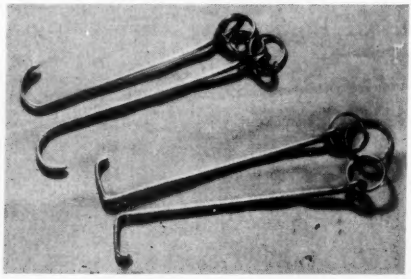

Fig. 2.—Two types of hooks have been used.

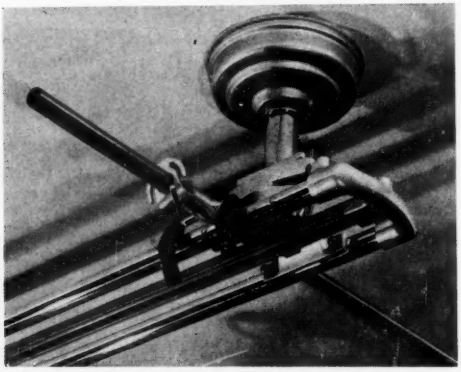

Fig. 3.—Method of fixation of rod to overhead lights.

is necessary. Then the hooks are inserted under the fifth ribs on either side of the sternum and the cord passed through a ring which supports the hooks. Traction is made on the cord sufficiently so that the sternum is elevated to the required amount. Two large clamps are placed on the cords for fixation.

Accidental contamination of the cord, prior to sternal retraction, occurred following manipulation of the overhead lights the first time it was used. This has been controlled by familiarity with the presence of the cord and delegating to one circulating nurse the complete responsibility for all manipulation of the overhead lights.